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# TRANSACTIONS

OF THE

## TWENTY-THIRD ANNUAL MEETING

OF THE

# AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY

HELD IN

ATLANTIC CITY, N. J.

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May 31st and June 1st, 1917

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OF THE

# AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY,

Held in Atlantic City, N. J., May 31st and June 1st, 1917.

## PRESIDENT'S ADDRESS.

By THOMAS J. HARRIS, M. D., New York, N. Y.

Fellows of the American Laryngological, Rhinological and Otological Society:—

In no mere formal words do I desire to express to you my deep appreciation of the honor which you have conferred upon me by electing me President of this Society. To be deemed worthy to follow in the footsteps of those who, in the past, have so ably discharged this office serves to fill one with humility and not with pride. It is as the result of their efficient labors that we can report to you today that the Society is enjoying in every respect unqualified prosperity.

Its membership is the largest in its history. Between thirty and forty applications are before your Council at present. We meet this year under conditions which, it is to be hoped, will never be repeated. The shadow of cruel war rests upon us as it does over every organization and every individual. It can be truly said however that the medical profession throughout it all has given its own life to save life, never to take it, and that however strongly we may feel regarding the political aspects of the war, we can confidentially look forward to no division when it is over in the ranks of science.

The founders of this Society defined its object "to be the promotion of the science and art of medicine, the development of its members in original scientific work and the promotion of social intercourse." Any one who has been a member for even a short time will testify to the delightful social intercourse which exists in

our ranks. One after another of the older members will bear witness to the truth of the statement that some of their warmest and most lasting friendships have been made through this organization. Properly conducted this will always be the case and the value of it cannot be overestimated.

We desire particularly to emphasize however the two other aims of the Society, namely the promotion of the science and art of medicine and the development of its members in original scientific work. Nothing is truer than that any Society that rests contented with what it has accomplished has completed the reason for its existence and had best disband. This Society in its twenty-two years of existence has given birth to some of the best work that has been done in otolaryngology. Our Transactions are abundant evidence of this, but we are capable of much more. Numberless problems affecting our specialty, many growing out of the war remain to be solved. This has impressed itself upon your Council the past winter with much weight and we have been concerned with the question how we can secure still better work from our members. It is recognized that the men who can do this are the young men who are not tied down by the exacting responsibilities which are bound to come with advancing years. It is these young men with the training which our schools give today whom we especially desire to add to our ranks and to whom we want to offer such opportunities for investigation that they can and will undertake it.

For many years the Society gradually has been accumulating a surplus which, in the mind of at least one of its members, has always been intended for some such purpose as this. At the present rate of growth however, it will be many years before it will reach the amount that will yield an income sufficient to secure this end. The Council accordingly had in mind to propose to you at this meeting that they be authorized to invite subscriptions from the members in larger or smaller sums to bring the present surplus up to the total of $25,000. It was their feeling that the income from this would be sufficient to maintain a scholarship and in that way attract one worker at least each year to the ranks of original scientific work. Closely in line with this proposed forward step is the recommendation from your Council which will come before you for consideration of the incorporation of the Society. As will be explained later this step for many reasons is desirable. If it receives your approval we can with confidence give now or later in our wills to the establishment of a permanent scholarship fund.

Alas, since the action of the Council in this important matter, the great cloud which has been hanging over Europe for so many months has reached our own dear land. We have been pulled into the maelstrom. All thoughts of our own advancement as a Society such as have just been outlined must be abandoned for the time being to be taken up again it is confidently hoped when we are once more at peace. For the present economy and retrenchment in all that concerns the financial affairs of the Society are encumbent upon us, and your Council will, with this in view, present at the proper time certain sweeping and what may seem at first radical recommendations for your consideration and approval.

Finally one word in regard to what is on the lips of us all at the present moment, namely what can we as medical men and specialists do to assist our country in its present crisis? Some few months ago, at the request of Dr. Martin of the Committee on National Defense, your President appointed a Committee to consider this matter. This Committee has given careful consideration to it and prepared a series of recommendations which they will present to you at the proper time and which will assist we feel in solving the problem for many of us. Already several of our members are at the front and others are going. Whether it is his duty to follow in their footsteps or to remain here each one of us will have to decide for himself. Wherever we are found however, of one thing your President is sure,—that one and all of us may be counted upon to do credit and honor to the medical profession and to this Society of which he is a part. It is now my pleasure to declare that the twenty-third annual meeting of the American Laryngological, Rhinological and Otological Society is open for scientific work.

# A PERSONAL STUDY OF SOME OF THE DISEASES INVOLVING THE MAXILLARY ANTRUM WITH SPECIAL REFERENCE TO THE NITRATE OF SILVER TREATMENT.

By JOSEPH H. ABRAHAM, M. D., New York, N. Y.

Diseases of the maxillary antrum are classified by modern authorities according to this scheme:

| | | |
|---|---|---|
| 1. | Catarrhal Inflammations | a. Acute<br>b. Chronic |
| 2. | Purulent inflammations | a. Acute<br>b. Chronic |
| 3. | Specific Inflammations | a. Syphilis<br>b. Tuberculosis, etc. |
| 4. | Neoplastic | a. Benign<br>b. Malignant |

This classification presents no novelty, but is simply a convenient guide in the description of the various lesions and is a convenient clinical aid, especially in regard to therapy. I shall emphasize especially the catarrhal, purulent and syphilitic inflammations.

The acute catarrhal form of inflammation begins with simple congestion, and swelling of the nasal mucous membrane. By direct extension into the antrum of Highmore or the respiratory tract a similar pathological lesion may be caused there. Swelling of the mucous membrane around the ostium causes retention with the classical symptoms and signs familiar to us all.

Less attention has been paid, however, to catarrhal inflammations due to metabolic or systemic conditions. In the various so-called diatheses the nasal mucous membrane may share in the elimination of irritative excretion with a consequent change, usually of a catarrhal type. The mucous membrane is swollen and hyperemic, and since there is vasomotor involvement, the swelling is evanescent, but may remain and become chronic. More often than in the acute catarrhal form from simple cold in the head or from local irritative factors, the mucous membrane instead of being hyperemic and swollen is pale and edematous. I shall cite a case in point. A young

college student of twenty-one years of age, complaining of frontal headaches and pains over both antra with a profuse watery nasal discharge, requiring from six to eight handkerchiefs a day, was examined by a prominent specialist in Chicago, and both maxillary antra and frontal sinuses were found on X-ray examination to give shadows. Operation was advised. The young man came to New York and consulted a second specialist who also advised operation. His father, a surgeon in the south, referred him to me. I recognized the condition as belonging to the toxic or metabolic group. The condition of the nasal mucosa gave me my clue, being pale and edematous. The boy's breath was fetid, and on transillumination both antra and frontals were dark. Upon inquiry as to his diet, I found that he was a very heavy meat eater. The urinary examination showed a urine of high specific gravity, a marked increase of indican and a large amount of uric acid. I ordered calomel, salts, a meat free diet, and the same local treatment as will be described under acute inflammations. In seventy-two hours the picture had entirely changed: the edematous condition of the nasal mucosa was relieved, the antra had discharged their retained contents, and the boy was and has continued to be free from all symptoms. I wish especially to emphasize that these cases are not uncommon, and operative interference predisposes to secondary infections. In these cases I advise a proper hygienic regime, in order to eliminate the metabolic disturbance. I regard the indicanuria and the increased uric acid excretion as evidence of this disturbance.

The acute catarrhal form may on account of the more intense nature of the inflammatory process develop rapidly into the acute purulent type, with involvement of the antrum with a resulting acute empyema of this cavity. The bacteriology of antral diseases is extremely complicated because of the fact that we usually get secondary invaders. It is true that we do get pure pneumococcus, bacillus mucous capsulatus, and influenza bacillus infections, also infections with hemolytic and an-hemolytic streptococci. However, in the more chronic types the predominant organism is the staphylococcus pyogenes. Again the organisms may be found secondarily in the presence of the acute exanthemata.

In the acute purulent type there is a marked redness, swelling with polymorphonuclear exudation with a resulting pus formation. As in the acute catarrhal form, the process here may resolve spontaneously or resolve under proper treatment, or it may become chronic. The chronic catarrhal form is due to repeated attacks of

the acute type. Simple chronic inflammations of the antrum are rare, but one does occasionally see a mucocele from increase in the secreting cells of the lining membrane. Retention of accumulated discharges of a mucoserous fluid with occlusion of the ostium causes a hydrops antra. According to Hajek, inflammatory tumors including cysts, polypi and hydrops of the antrum are the result of chronic inflammatory changes in the mucous membrane.

In the chronic purulent form, the mucous membrane thickens markedly and becomes pyogenic, and there is connective tissue proliferation. Polypoid masses may occur in the antrum. There is very marked pus formation. Again, and I desire to lay great emphasis on this point, there may be a direct extension of the infection from the roots of the molar teeth to the antrum. The presence of dead molars from nerve extraction and faulty fillings or a pericementitis with a small necrotic area at the tip of the root, the apical abscesses and the erosion of the alveolar process characteristic of pyorrhea, may by contiguity involve the malar bone and cause an osteomyelitis, which lesion later involves the floor of the antrum. Pus accumulates in the antrum and we are now dealing with a typical case of chronic purulent empyema. In fact, about 50% of the cases of chronic empyema of the antrum of Highmore that came under my observation, were due to tooth complications. Necrosis of the bone in the antrum, which is so dreaded by rhinologists and warrants their performing the radical operation in so many cases, is due either to root infections or to syphilis. Personally, I have never seen a case due to any other cause. Therefore, I should advise in every case of chronic empyema of the antrum in which the nerves of a molar tooth have been extracted or destroyed, whether pain in the tooth is present or not, to have an X-ray examination made immediately.

### *Syphilitic Empyema.*

In November 1905, I read a paper before the Section of Laryngology of the New York Academy of Medicine, on "Syphilitic Empyema of the Accessory Sinuses of the Nose, with a Report of Four Cases." Since then our literature has very little to say on this very important subject. Our modern text books devote pages in describing syphilis of the nasal cavities, but only a few lines to syphilis of the accessory sinuses. I will quote the following from Kyle's modern book. "Syphilitic affections are of possible location in the antrum, but are extremely rare and when so occurring are con-

comitant with the nasal lesion of the same character." With our modern aid in diagnosing syphilis, all cases of intranasal lesion of a doubtful character should be given the benefit of a Wassermann test.

Since publishing the above article, I have treated nine cases of syphilitic sinusitis. The time allotted will not allow me to describe in detail the pathologic lesions. In the near future it is my intention to report these interesting cases.

In view of the remarkable advances in roentgenology we are able today to add the corroborative evidence of the X-ray plate to our other aids in diagnosis. The plates reveal a cloudiness in the region of the antrum and also of the other sinuses if these too are involved. Differation between a serous and purulent involvement, however, is dependent upon suction, puncture and the finding of either mucus or pus in the cavity.

*The Treatment of Acute Cases.*

Should be local and internal. If the patient has temperature and climatic conditions are disagreeable, order him to bed and administer broken doses of calomel and soda followed by saline. A capsule containing the following should be administered every three hours, as in my experience it reduced congestion of the nasal mucosa and relieves pain.

| | | |
|---|---|---|
| Rx. | Hyoscine Hydrobromide | gr. 1/150th to 1/250th |
| | Camphor Monobromated | gr. ss |
| | Quinine Hydrobromide | gr. 1ss |
| | Sodium Bromide | gr. V. |

A diet free from all meats, sweets, tea, coffee and alcohol is ordered. Locally, I advise steam inhalations through the nose. These should always be given in bed, never in the doctor's office. Hot electric or water bag or electric light baths may be given to control local pains. I further advise in order to promote better drainage of the sinus the following ointment:

| | | |
|---|---|---|
| Rx. | Cocain Alkaloid | gr. V. |
| | Ichthyol | gr. XL. |
| | Chloretone Inhalant Special* | ʒIV. |
| | Adrenalin Ointment | ʒIV. |

M. Ft. Ungtt. in collap. tube.
This ointment melts at 98° F.
*The same formula as Parke, Davis' preparation, substituting olive for the mineral oil.

This has proven wonderfully efficacious in my hands, and is applied to the region of the middle and superior meati and turbi-

nates by the doctor, and three or four times a day the patient should apply this ointment as far into the nasal cavities as possible. Negative pressure or suction followed by positive pressure and medication of the sinus as advocated by Dr. Lewis Coffin is of great value in these cases.

If pain persists after two or three days treatment, irrigations of the antrum with a sterile normal salt solution through the inferior meatus must be resorted to. Cocainize the inferior meatus and with my trocar needle the antrum is punctured about 1.5 to 2 c. c. posterior to the anterior attachment of the inferior turbinate. A few irrigations are usually sufficient.

In acute purulent inflammation the treatment is about the same. A few cases will persist and in these an antiseptic astringent solution is indicated. After experimenting with various agents for over fifteen years, I have obtained my best results with solutions of nitrate of silver. A detailed description of this technique will be given under chronic empyema.

*Treatment of Chronic Empyema.*

In March 1907, I presented a paper before the Section of Laryngology and Rhinology of the New York Academy of Medicine on "The Indications and Advantages of the Intra-Nasal Over the Radical Operation in the Treatment of Chronic Empyema of the Antrum of Highmore and the Technique to be Employed," reporting thirty-four successful cases treated by me beginning in 1897. Since 1907, I have treated several hundred cases successfully. The only change in treatment was in the direction of simplifying the technique. It affords me great pleasure to present to you the technique and treatment now employed by me.

In simple chronic empyema apply cocain and adrenalin to the nasoantrum wall, inferior turbinate, floor and the adjacent surface of the septum. Resect the lower border of the inferior turbinate with Struyken-Abraham forceps down to the bone and if necessary remove a small portion of the lower border of the bone for about 3 c. c. After washing the antrum by means of my needle and syringing through the inferior meatus, cocainize the antral-mucosa. Then enlarge this opening with my antrum reamer and burr. The antrum should be opened about 1.5 to 2 c. c. from the floor of the nasal vestibule in order not to injure the lacrimal duct. If granulations are present remove them with a Myles curet. Never remove the mucous membrane. After thoroughly douching the an-

trum and after all excess solution is drained, the nasal vestibule is plugged with cotton, which filters the air and prevents a sudden dilatation of the blood vessels. On the following two or three days the antrum is irrigated with a normal saline solution. About the third day begin the nitrate of silver treatment. Spray the nasal mucous membrane with a 2% solution of cocain. Irrigate the antrum through my silver canula with plain sterile water. Insert a piece of absorbent cotton over the antrum opening and spray the nasal mucous membrane with liquid alboline. Remove the cotton, apply vaseline over the face thoroughly, then insert the silver canula into the antrum, and bend the patient's head sideways with a slight inclination of his face downwards, and forward so that the diseased cavity will be lowermost. Syringe from 2 to 5 c. c. of a weak solution of nitrate of silver, usually a 1% solution, into the antrum. My idea is to apply the silver solution to the entire mucous membrane covering the antrum. The excess should remain in the cavity for about a minute, then reverse the patient's head and allow the excess to drain. To avoid excessive sneezing cover the antrum opening again, and spray the nasal cavity with a normal salt solution of chloretone inhalent. Dry the patient's face of vaseline and plug the nasal vestibule. This plug should be changed often by the patient as the silver stimulates the entire nasal and antrum mucous membrane. Quoting verbatim from my original article as follows, "Day by day the strength of this solution (nitrate of silver) is increased until the mucosa assumes a normal appearance." This requires from ten days to three weeks, leaving your patient the possessor of a perfectly normal antrum with a minimum destruction of the inferior turbinate and nasal mucosa. Furthermore, the action of the silver salt on the nasal membranes, relieves congestion, destroys infection, reduces hypertrophy and your patient is cured of his so-called "Nasal Catarrh."

In my series of cases, a 20% solution of nitrate of silver was the strongest used. Since my original article, Roe[1], Hurd[2], and others, have reported good results in the use of nitrate of silver. Hurd in some cases employs the saturated solution. I have never seen a case requiring so strong a solution.

In cases of chronic empyema, due to or complicated by root abscess, with necrosis, I prefer a general anesthesia. A dentist extracts the offending foreign body, and with oval shaped curets all of the necrosed bone up to and within the antrum surrounding

1. Roe—Annals of Otol. Rhin. & Laryn. June, 1909.
2. Hurd—Annals of Otol. Rhin. & Laryn. Dec., 1911.

this region is curetted through the tooth socket. All granulations are removed with Myles' malleable curets. Intranasally the same operation as described under chronic empyema is performed, with the exception that this opening is made a little larger and nearer to the floor of the nasal cavity. Unless hemorrhage is severe no packing is placed into the tooth socket. Very often I suture the opening with silk. This should be allowed to close as soon as possible, and in fact it is better to wait a few days before beginning the nitrate of silver treatment through the nose, so as to get a complete or partial closure of the alveolar opening.

In two cases owing to the great amount of bone removed from the alveolus a fistula persisted for several weeks. This I overcame by freshening the edges of the fistula, and cutting through the soft tissue and periosteum down to the bone, 1 c. c. inwards and parallel to the fistula, elevating the soft tissues and closing the tract with several silk sutures.

The treatment of syphilis should be general as well as local. Locally, all that is necessary, when the gumma is broken down, is to secure thorough drainage. This is accomplished in the manner described under chronic empyema. It is far better to be conservative and await the result of general anti-syphilitic treatment on the local lesion, than to do more radical work. Subsequently, if necrotic bone is present, this must be removed. In gummas involving the nasal cavities, a Herxheimer reaction is not uncommon, so one must begin with small doses of salvarsan, about 0.2 gram.

Quoting from Loeb's "Operative Surgery of the Nose, Throat and Ear," 1917 Edition, Volume 2, on "'The Results of Oral Operations on the Maxillary Sinus: Various wound accidents and other unpleasant results may follow this operation. Among them may be mentioned: Aspiration pneumonia, anesthesia of the cheek or teeth, emphysema of the cheek, neuralgia of the fifth nerve, stenosis of the nasal duct, and osteomyelitis of the maxilla." I maintain that all these unfortunate accidents can be avoided by the methods described by me.

In conclusion, I can only add after many further years of experience with this technique, that conservatism has been my watch-word, and I meet the surgical indication as it arises rather than perform many radical and frequently unnecessary operations.

DISCUSSION.

**Dr. Ross Hall Skillern,** Philadelphia, Pa., was impressed with the remarkable divergence of view at the present time with regard to the frequency of maxillary sinusitis of dental origin. Dr. Abraham had

given it as fifty per cent.; others had stated it as being all the way from two to one hundred per cent. In his own experience it was about twenty-five per cent.

It was to be recalled that maxillary sinusitis might very readily retrograde, and infect the roots of the teeth, so that it might be said that the sinus infected the teeth, rather than that the teeth infected the sinus.

He had had no experience with dietetics in the management of these cases. Irrigation was rather a trite proposition, inasmuch as it was the practice of the majority to make a needle puncture and to combine the needle puncture with therapeutic measures, continuing to irrigate, after making the puncture, beginning with normal salt solution, and working up to nitrate of silver. It had been shown that it was not futile to carry a patient over a number of weeks, finally getting a cure. A French writer had reported that only after fifty-three irrigations was he able to cure a maxillary sinusitis. Dr. Chamberlin had reported making twenty-three irrigations in one of his cases, the patient having refused operation.

Resection of the inferior turbinate, which Dr. Abraham had included as a part of his treatment, was not necessary. Many operators had given up the sacrifice of any tissue in the treatment of maxillary sinusitis, removal of the pre-maxilla being sufficient. An incision could be made in front of the inferior turbinate and the sinus inspected.

**Dr. Harris P. Mosher,** Boston Mass., was in accord with Dr. Skillern in the matter of the treatment of diseases of the maxillary antrum. Opening the antrum, plus aeration, was sufficient, irrigation being necessary in only a small proportion of cases. He was greatly surprised to hear the essayist report seven hundred cases of disease of the maxillary antrum, even from a city like New York, and was rather inclined to think he had confused acute with chronic cases. Had he persisted in the treatment of acute cases he would probably have obtained just as good results as those reported in the paper. It was also surprising, that in one or two acute cases Dr. Abraham had waited so long before washing out the antrum. It was not apparent why he had not put in the needle the first day instead of waiting for general medical treatment. It was equally difficult to understand why he waited until he had had the teeth X-rayed, why he did not have them X-rayed at the first examination, and why, in some cases, he waited for the removal of the teeth. In the speaker's experience the removal of the teeth cleared out the antrum.

As to operative procedure, he had never had faith in or liking for the intranasal treatment of suppuration of the maxillary antrum. He had always felt that any intranasal operation gave nothing more than a peep-hole into the antrum. If a case did not clear up under irrigation within six weeks it had been his custom to employ the old-fashioned canine fossa operation, with a large opening into the nose. Having done this, he had never found it necessary to resort to any special form of irrigation.

**Dr. Lee M. Hurd,** New York City, recalled a case in which there was involvement of the frontal sinuses, the ethmoids, and the maxillary antrum, with abscess of the orbit, in which the infection had extended through the roof of the antrum. He had never operated in an acute maxillary sinusitis. He had always used the needle early, washing out with normal saline solution, and, if one or two washings did not clear up the condition, had filled the antrum with bismuth paste, repeating this every other day. It was a painless procedure, and cleared up the cases promptly. Some of the patients had drifted away with the antrum filled with paste. He saw no reason whatever for opening the antrum through the canine fossa. He had abandoned the method, except when doing a Killian operation upon the frontal sinus. Drainage

was much more quickly accomplished through the inferior antral wall. In dental cases not only must the teeth be removed, but the bone must be curetted. It was possible, with nerve-blocking, to operate with perfect safety and comfort without the disadvantage of general anesthesia.

**Dr. J. W. Jervey**, Greenville, S. C., was glad to hear Dr. Myles emphasize the importance of getting down to something basic, and to hear Dr. Abraham refer to acute and chronic catarrhal inflammation, thus bringing before the Society the question of a definition of the term, catarrhal inflammation. It was as easy to talk about a catarrhal inflammation as about rheumatism, heart failure, senile decay, or any of the other vague terms in use. One knew that inflammation might be acute or chronic, passive or active, due to heat, chemical action, or vasomotor disturbance, or to some vis a tergo. In which category would one place catarrhal inflammation? Pathologically speaking, perhaps, any inflammation of a mucous membrane might be spoken of as catarrhal, but that was a question. In the class of catarrhal inflammations, would Dr. Abraham make irrigations per via naturalis, or by puncture? The speaker would do neither.

**Dr. Joseph C. Beck**, Chicago, Ill., had expected, from the title of the paper, to see a demonstration of pathological specimens, and was disappointed to find in it no exposition of real work on the pathology of the maxillary antrum. He had especially anticipated seeing some histological specimens of syphilis of the sinus, inasmuch as Dr. Abraham had called his attention to the fact that he was the first to demonstrate syphilitic sinusitis. It was recalled that in a paper which the speaker had presented before one of the sections of the New York state meeting, reference was made to the rarity of this condition. Dr. Abraham had then given him a reprint dealing briefly with the subject. This contribution of Dr. Abraham's was not indexed, which was unfortunate, as a question of this kind should be published at length. The importance of the teeth in relation to the diagnosis of disease of the maxillary antrum had been clearly demonstrated by Dr. Haskin. Bismuth paste was useful only in certain cases of chronic sinusitis. When the pathological process involved the mucous membrane there was nothing to be done except an obliteration operation.

**Dr. Abraham**, in closing the discussion, called attention to the fact that in the time allotted it was impossible to cover completely such a wide field, which perhaps accounted for some of the misunderstandings on the part of the speakers in the discussion. The relative frequency of dental cases, which he had given as fifty per cent., and which Dr. Skillern had found to be twenty-five per cent., was doubtless the outcome of a difference in experience. Dr. Mosher had expressed surprise at the number of cases reported; the number was placed at "several hundred," not "seven hundred," as Dr. Mosher had understood. As an illustration of the frequency with which these cases presented themselves in the speaker's experience, he recalled that during the last five days he had sent three cases of chronic empyema of the maxillary antrum to Dr. Caldwell for X-ray examination, the diagnosis being verified in each case. Another misconception was with reference to the technic. He did not remove the anterior end of the inferior turbinate, but merely dissected away the inferior border. When these cases healed they had perfectly normal mucous membrane. In acute cases he used mild solutions; and he had specifically stated that in some cases he employed no irrigation at all. His purpose always was to restore to the patient a normal antrum, which it was possible to do.

The teeth might become infected on the opposite side, the teeth on the same side as the antral infection not being involved. He agreed

with Dr. Hurd with regard to the frontal sinus and ethmoid cells. The key to the entire situation was the question of drainage. Necrosis and granulations must be removed. The tendency of the opening to close, as suggested by Dr. Myles, would be largely obviated by making the opening as near to the floor as possible. He had had no experience with bismuth paste, as suggested by Dr. Hurd, his experience with nitrate of silver having proved entirely satisfactory. The method, proposed by Dr. Myles, of opening through the hiatus semilunaris, did not always give satisfaction.

Answering Dr. Jervey, the question of classification had been thoroughly covered in the paper.

He expected, at a future time, to publish the pathological data, the lack of which in the present communication had disappointed Dr. Beck.

# REPORT OF A CASE OF COEXISTENT CARCINOMA, TUBERCULOSIS, AND SYPHILIS OF THE ESOPHAGUS.

By L. W. DEAN, M. D., and J. B. GREGG, M. D., Iowa City, Iowa.

The purpose of this article shall be to briefly discuss carcinoma, tuberculosis, and syphilis of the esophagus; their coexistence in this organ; with the report of a very unusual case.

## Tuberculosis of the Esophagus.

The first case of esophageal tuberculosis was recorded by Denonvilliers (1) 1837, and six years later Louis reported finding six cases of esophageal ulceration, presumably tuberculous, in 120 tuberculous cadavers.

In the seventy-four years which followed this first observation, 103 cases were reported; only 69 of which, can, with reasonable certainly be classed as tuberculous. Since then, numerous cases have been reported, until now the affection is no longer regarded as being of great rarity.

This disease has, however, received such slight consideration in medical literature, that the physician does not take it into consideration in an otherwise tuberculous patient and may ascribe the esophageal symptoms to a visible laryngeal tuberculosis.

The classification according to etiology, as suggested by Flexner (2) is as follows:

1. Instances in which the tuberculous process arises through continuity or contiguity of structure; as in ulceration of the esophagus from a caseous bronchial gland, caries of the vertibrae, or from a tuberculous ulcer of the pharynx which passes down and invades the esophagus.

2. Cases in which there exists in the esophageal mucosa a previous lesion to be regarded as predisposing to tuberculous infection.

3. Instances in which (a) the esophagus is affected in the course of a general disseminated miliary tuberculosis; and (b) in which there is infection of the mucosa from tuberculous sputum where no previous lesion existed.

Of the nineteen cases of undoubted tuberculosis collected by Flexner, but two cases belonged to Class 2, in which we are especially interested in this report.

When seen as an extension from the larynx, especially as "party wall" lesions, the esophageal lesions present much the same picture as the laryngeal disease (3). When, however, they are not confluent with a lesion in the pharynx or larynx in the esophagus, the endoscopic appearances are those of superficial ulceration, or a simple erosion, and there may be yellowish or whitish granules in the neighborhood of the erosions or ulceration. Cicatrices have been observed by Jackson but twice; once in connection with ulceration; and once in invasion of the esophagus by peri-bronchial tuberculous grandular processes. The ulcerations of esophageal tuberculosis usually are more superficial and less inflammatory in character than either cancer or lues. The diagnosis here as elsewhere rests upon the microscopic findings in pieces of involved tissue removed by means of the esophagoscope.

## Syphilis of the Esophagus.

Syphilis involving the esophagus, although rare, has long been known to exist, having been described by Marcus Aurelio Severino, (1580-1656), (4). Prior to the days of esophagoscopy, (5), the few diagnoses of lues of the esophagus were made solely from the disappearance of esophageal symptoms following antiluetic treatment. Such diagnoses necessarily are apt to err, (6).

The esophagoscope has shown that syphilis of the esophagus may present itself as the mucous plaque, a gumma, an ulcer or as a cicatrix. For diagnosis, the esophagoscopic appearance must be considered in relation to the history, the concomitant lesions, the therapeutic test, the Wassermann reaction, the examination of the tissue, and the search for spirochaetes.

The esophagoscopic appearance of the luetic ulcer is largely due to mixed infection, with resultant inflammatory condition. The mucous plaque appears similar to that seen in the fauces. It may be slightly elevated in one part, with simply a bluish white cloudiness in another part. The lesion is typically inflammatory in character. The cicatrix of luetic esophagitis does not differ from other cicatrices (7).

## Carcinoma of the Esophagus.

Carcinoma is the most important neoplasm and the most common disease affecting the esophagus.

Eighty per cent. of malignant esophageal cases are carcinomatous (8).

In 5,079 autopsies, Zenker (9) found carcinoma of the esophagus primary in only thirteen cases; and secondary in but six. Williams (10) has found them to constitute 5.3 per cent. of all carcinomatous tumors, and to be much more frequent in men than in women. Von Hacker, (11) states that over 72 per cent. occur in males, and that the neoplasm is usually primary in the esophagus. They may develop by extension or by implantation (12), but do not occur in this locality as the result of metastasis (13).

Carcinoma of neighboring organs may spread to the esophagus and be inoculated from a cancer situated higher up or lower down, and the entire tube may become covered with carcinomatous nodules. The cancer is usually an epithelioma (pavement epithelium); glandular cancer and the colloid form are more rare. As a rule, only a portion of the esophagus is attacked, with the production of insular infiltration forming a segment on one side of the tube, or annular, cauliflower-like and papillomatous tumors.

Carcinoma of the esophagus is found most frequently in one of three localities at the cardiac orifice, opposite the bifurcation of the trachea, or on a level with the cricoid cartilage.

Jackson gives the following forms of esophageal carcinoma:

1. Submucosal infiltration covered by perfectly normal membrane; usually associated with more or less bulging of the esophageal wall, and usually associated with hardness and infiltration
2. Leucoplakia.
3. Ulceration projecting but little above the surface at the edges.
4. Rounded nodular masses grouped in mulberry like form either dark or light in color.
5. Polypoid masses.
6. Cauliflower fungations.

It is always well to remember that a carcinomatous lesion which has existed for some time may have engrafted upon it a mixed infection with resulting inflammation.

The real diagnosis rests on the histological examination of a specimen removed esophagoscopically. Submucous tumors should not have a piece removed for diagnosis because of the danger of causing ulceration, and rapidly increasing the growth of the tumor if it be malignant.

Seventy per cent. of the cases of esophageal carcinoma have been found in patients of above fifty years, but it has also been observed in young individuals (19 years of age).

Irritation, mechanical, chemical or thermal, is a most important predisposing factor in the etiology (14).

Zenker believes that alcohol is a very common irritant to the esophageal mucosa and may predispose to carcinoma.

Körner (15) and Voelker attribute most esophageal carcinomata to the irritation produced by the passage of large boluses of poorly masticated food and this has been in accordance with the apparent etiology of the cases, with which we have dealt.

It is doubtful if any antagonism exists between carcinoma and tuberculosis; there is also no special favoritism; yet cancer is more frequent among those with latent tuberculosis than among those at the cancer period of life (16).

The coexistence of tuberculous disease and cancer in the same part has been shown by Crawford, in the mammae; Franque, in the uterus; Frerichs, in the liver; Beale (17), in the larynx; Dalton, in the large intestine; Naeghi, in the rectum, ilium and caecum; Dixon (18), in the intestine, and by Lapeyre (19), in the hand.

We recently had a case of cancer of face and esophagus. In our judgment each was primary in an individual predisposed to carcinoma.

Certain observers have held that the tubercle bacillus and its toxins are concerned in the etiology of cancer, but Dixon, Smith and Fox (20) after animal experimentation convinced themselves that the tubercle bacillus and its products could stimulate epithelial structures to hyperplasia, but not produce an invasive tumor. To explain the combination of the two diseases, two theories have been suggested (21):

First, that the chronic inflammatory action of tuberculosis prepared the way for the development of cancer.

Second, that cancer by lowering the general nutrition, assisted the growth of tuberculosis.

Cases of coincident carcinoma and tuberculosis of the esophagus have been reported (22).

Zenker (23) reports a case of stricture of the tube, produced by a girdle ulcer of rodent character, which on examination microscopically proved to have the typical appearance of a squamous epithelioma. The growth invaded the deeper strata of the mucosa and at times penetrated the muscularis. In the border of the cancerous portion, near the normal tissue, typical tubercles existed, some of which were caseous. The neighboring lymph glands were also involved in the cancerous and tuberculous conditions. Tubercle bacilli were found in small numbers in the tubercles. Zenker

favors the view that the carcinomatous lesion was primary, the intact epithelium being resistant to tuberculous infection.

Cordua (24) reports the case of a man of sixty years, who came to autopsy with the clinical diagnosis of carcinoma of the esophagus, incipient pulmonary tuberculosis and adhesive pleuritis. In the esophagus, at about the bifurcation of the trachea, was found an ulcer ten to fifteen cms. long, which affected the whole circumference. The edges of the ulcer were somewhat elevated and firm, the base was soft and pulpy. Upon sectioning the ulcer, the entire wall of the esophagus was found almost destroyed forming a small tumor mass. Microscopically, both a carcinomatous and a tuberculous lesion was found. Tubercles in the process of caseation were found in the middle of the cancerous ulcer, and about its periphery. No tubercle bacilli were found after careful search.

Cordua thought the tuberculous invasion of the cancerous ulcer took place through the lymphatics leading from tuberculous areas in the lungs. It would seem, however, more probable that the secondary tuberculous involvement was due to sputum deglutition.

Pepper and Edsall (25) (26) report the case of a man, forty-two years of age, who had tuberculosis of the lungs, larynx, cervical glands and esophagus. The esophagus was patulous and normal as high up as the level of the arch of the aorta. Above this point and to the junction with the pharynx, the lumen had entirely disappeared.

The esophageal walls and surrounding tissues had formed a band of dense fibrous tissue about 1.25 cm. thick and 3.75 cm. broad which was firmly bound to the vertebrae. Examined microscopically, cancerous tissue was found on a dense fibrous tissue basis; a tubercle like mass undergoing fibroid changes, with an occasional giant cell, and tubercle bacilli.

The central and deeper portions of the tumor were composed mainly of fibrous tissue; this leading the authors to conclude that tuberculosis was the original cause of the disease, while cancer subsequently infiltrated the occlusion.

Weyeneth (27) reports the case of a man of fifty-eight, who had pulmonary tuberculosis for five years, for four years suppuration and deafness of the left ear; and later involvement of the right ear. At the time of examination there had recently developed difficult deglutition and vomiting after eating. A sound was passed into the stomach without meeting an obstruction. After three weeks, death occurred; the cause being given as "tuberculosis pulmonum et in-

testini." The autopsy showed tuberculosis of both lungs and several ulcers in the neighborhood of the ileocaecal valve. On the anterior wall of the esophagus, and on a level with the bifurcation of the trachea, was an oval ulcer that macroscopically gave the impression of an ulcerated tumor. Surrounding the ulcer was an area over which the mucosa was destroyed. Scattered throughout this denuded area were numerous small nodules, both isolated and grouped. Granulation tissue covered the base of the ulcer, and the esophageal wall in the neighborhood of the ulcer was thickened. Microscopically the chief mass was found to be composed of cancer tissue with recent tuberculosis; tubercle bacilli being found in the nodules about the periphery.

In this case, the sequence was: tuberculosis of the lungs and intestines; carcinoma of the esophagus; tuberculosis of the esophagus. Weyeneth's opinion, concurred in by Professor Ribbert, was that the malignant growth antedated by several months, the tuberculous invasion. Infection by the sputum (28) seems probable as there were no foci in the neighboring lymph glands.

Syphilis in combination with carcinoma or with tuberculosis, is more rare.

Kellert (29) has found relatively few cancer patients, who gave a specific history, and Wassermann tests on their blood serum seldom gave a positive reaction.

Occasionally (8) leucoplakia has been observed in the esophagus similar to that of other mucous membranes, coincident with carcinoma of the esophagus. As carcinoma develops in other situations where there is leucoplakia, it is possible that here also it may occur with in some cases of syphilis as the causal agent for the leucoplakia.

According to Lexer Bevan (30) carcinomas of the mucous membranes may develop from fibro-epithelial growths, and from ulcers of all kinds, especially the tuberculous and syphilitic.

Dennis (31) has notes on a case in which a diagnosis of tuberculous laryngitis was made in a woman with a history of pulmonary and hip joint tuberculosis. Later a characteristic skin syphilid was discovered, and under antiluetic treatment the larynx rapidly healed, except in a comparatively small area, which remained obstinate. He considered that this was probably a case of syphilis of the larynx on which a tuberculous infection was grafted.

According to Dennis (31) Moritz Schmidt has reported a case in which tuberculosis, syphilis and carcinoma were all present in the larynx of the same patient.

Other writers (32) (33) report laryngeal cancer having coexisted with laryngeal syphilis and with laryngeal tuberculosis.

Jackson (34) reported a case before this Society, in which he was certain that carcinoma, syphilis and tuberculosis coexisted in the larynx. The patient, a man of fifty-two, had a family history of tuberculosis, a history of a primary luetic lesion three years before, followed by well marked secondaries, and at first examination, a gumma of the septum. The larynx was in the ulcerative stage; the respiratory chink being narrowed to an oblique slot. Fungating masses protruded from the ulcers, but not sufficiently to completely occlude the laryngeal lumen. The neck was full of enlarged hard lymphatic glands. Jackson considered the diagnosis of laryngeal syphilis with perichondritis indisputable, and the patient was put on vigorous mixed treatment, with marked laryngeal improvement. Healing occurred until the only lesion was a fungous, warty-looking mass on the left cord and filling the left ventricle. Here all improvement ceased and overwhelming doses of potassium iodide, with mercury to full tolerance, for two months, had no effect on the growth which began slowly to increase in size. Repeated examinations of the sputum revealed tubercle bacilli. A specimen of the larynx was removed, and proven to be an epithelioma.

Kellert (29) reports the case of a woman whose post mortem examination led to the following anatomic diagnosis:

Carcinoma of the pharynx with metastases to the cervical lymph node; tuberculosis involving the same area of the pharynx; tuberculosis of the same lymph nodes as involved by the carcinoma; pulmonary tuberculosis with cavity formation; tuberculous pleuritis with effusions; tuberculous ulcers of the small intestine; and tabes dorsalis, syphilitic.

Moritz Schmidt (35) has observed a case with Roth, which was diagnosed tuberculosis of the larynx. There was healing of this lesion, yet in spite of the fact that the patient denied syphilis, tertiary lesions were found in the larynx. There was also partial healing of this second lesion, but a growth diagnosed microscopically by Weigirt as carcinoma, appeared upon this site, and from which the patient finally died.

CASE REPORT.

*Case.* Mr. J. S., white, male, sixty years of age, german, a tailor.

Referred by Doctor F. W. Bailey of Cedar Rapids, Iowa.

The patient entered the University Hospital, April 17, 1916, complaining of inability to swallow well, weakness and cough.

*Present Illness.* Twenty-eight years ago he first had trouble in swallowing, lasting for five weeks. His only trouble at that time was dysphagia; no loss of weight; no cough; no regurgitation or lodging of food. This attack began suddenly and ended suddenly without treatment of any kind. Since that time up to November, 1915, he had no trouble of any kind.

In November, 1915, while eating supper, he swallowed a piece of meat not well masticated; which seemed to lodge just back of the sternum, causing violent coughing. The coughing spell soon ended and the feeling as of something lodged in the throat disappeared. He drank some liquids at once without difficulty, and the next morning he ate as usual.

For about six weeks he swallowed as well as ever, but about the last of December, 1915, he noticed that he was becoming poor. About January 1, 1916, he began again to have some trouble in swallowing; the amount of dysphagia varying considerably. Gradually the swallowing became more difficult so that articles of solid food were not eaten. Even in swallowing soft food he would often choke and cough so violently that food was expelled through both the nose and the mouth. He coughed a good deal, but not at any particular time; no event seemed to bring on these attacks. No blood was ever coughed up; only a very tenacious slimy sputum, like the white of an egg only more sticky. He does not regurgitate food eaten hours before. The throat feels dry and he has marked thirst; which owing to the difficulty in swallowing he cannot satisfy.

At the present time he is unable to swallow anything with surety. He has lost fifty pounds in weight, and is markedly weakened, sallow and anemic. He complains of pain in the chest just behind the sternum, which is severe enough to awaken him at night. During these attacks of pain he finds it difficult to breathe.

*Family history.* His father and mother are dead; the cause of death being unknown. He has five half brothers alive and well; one half sister alive and well.

He has four children alive and well. His wife has had three miscarriages, and one still-birth.

*Past history.* He had measles as a child, but no other childhood diseases; no fevers with the exception of malaria.

He uses beer and whisky moderately; never to excess. He chews tobacco moderately. He denies venereal history, but in 1887 he had a swelling of the cervical glands and a very sore throat. No other glands became enlarged, and there was no skin eruption. He denies sweats and hemoptysis; has had some cough, but not excessive. He catches cold easily but there has never been any dysponea.

He was hit on the head at one time, resulting in a skull fracture which caused the left ear to become deaf.

*General examination* by Doctor C. P. Howard, May 5, 1916.

The patient is a poorly nourished and developed man of stated age and gives the appearance of having lost much weight. The temporal arteries stand out prominently.

The muscles are small and there is a marked myoidema. The skin is hot, moist and covered with acne; without scars or areas of pigmentation suggesting a previous lues.

The thorax is of the senile emphysematous type, with high clavicles and rounded back. Expansion is fairly restricted equally. Litten is present on both sides. The percussion note is definitely impaired in the right supraspinous fossa and over the right clavicle. No changes in vocal fremitus. The breath sounds are feeble and vesicular; nowhere prolonged. Definite fine crepitations are heard over the apex, spine, clavicle and scapula on the right side; an occasional crepitation being heard over the left scapula.

*Heart.* The point of maximal intensity is in the fifth interspace, 8 cms. from the mid-sternal line. The area of relative cardiac dullness extends 4 cms. to the right of the mid-sternal line and 7.5 cms. to the left. The heart sounds are distant; no murmurs; A2 is not definitely accentuated.

The radial pulse is synchronous and equal; of good volume.

The blood pressure is systolic, 118 mm. Hg.; diastolic, 60 mm. Hg.

There is no tracheal tug and no retrosternal dullness.

The abdomen is flat and doughy; no masses or areas of dulness. The liver or spleen cannot be felt.

The shins are free from edema. There are two or three nodes over the right shin; possibly traumatic. The reflexes are normal on each side.

*Diagnosis.* Tuberculous involvement of both apices.

*Routine examination.*

*Eyes.* Chronic catarrhal conjunctivitis is present, with some lacrimation; tear ducts patent. The pupils are round, equal, 2 mm. in diameter, reacting to light and accommodation. Rotations are normal. The media are clear; the fundi showing no evident lesion.

O. D. V. = 6/60
O. S. V. = 6/60

*Ears, right.* The canal is clean; drum retracted slightly, but the ossicles move with Siegle's otoscope. High notes are well heard, but low notes are slightly cut down. He hears A C3 and C4 tuning forks; not the A fork. Weber's localized to the right ear.

*Left.* The canal is clean, drum retracted, the ossicles not moving with Siegle's otoscope. Galton whistle and none of the tuning forks are heard by air.

Hearing with alarm: Right—Whispered voice, 12 feet; spoken voice, 45 feet. Left—Whispered voice, 0; spoken voice, 0.

*Nose.* The septum is deflected to the right, with a large spur on the left. Each middle turbinate is enlarged. There is a small amount of mucopus in each middle meatus.

*Throat.* The teeth are in very poor condition, with marked pyorrhea. The pharyngeal mucosa and posterior buccal mucosa are markedly reddened. The lower pharynx and larynx are covered by a profuse amount of mucus.

*Esophageal examination.* On swallowing liquids, he coughs and chokes, nearly all the fluid being ejected. No fluid can be heard entering the stomach, and there is no gurgling in the neck. Choking comes immediately after swallowing.

The radiogram taken after swallowing bismuth and acacia mixture shows an S shaped stricture of the esophagus; the convexity of the upper portion is to

the right, of the lower portion, to the left. The middle of the stricture is at about the union of the manubrioclavicular articulation. The stricture apparently begins at the level of the lower margin of the cricoid cartilage.

FIGURE 1.

Photomicrograph of the tuberculous portion. Note especially the large typical giant cells.

April 21, 1916, the patient was esophagoscoped under cocain anesthesia, using the Mosher and Jackson esophagoscopes.

One and one-half inches below the cricoid cartilage, was found a nodular mass on the right side of the esophagus. The lumen of the tube was very small and to the left of this mass.

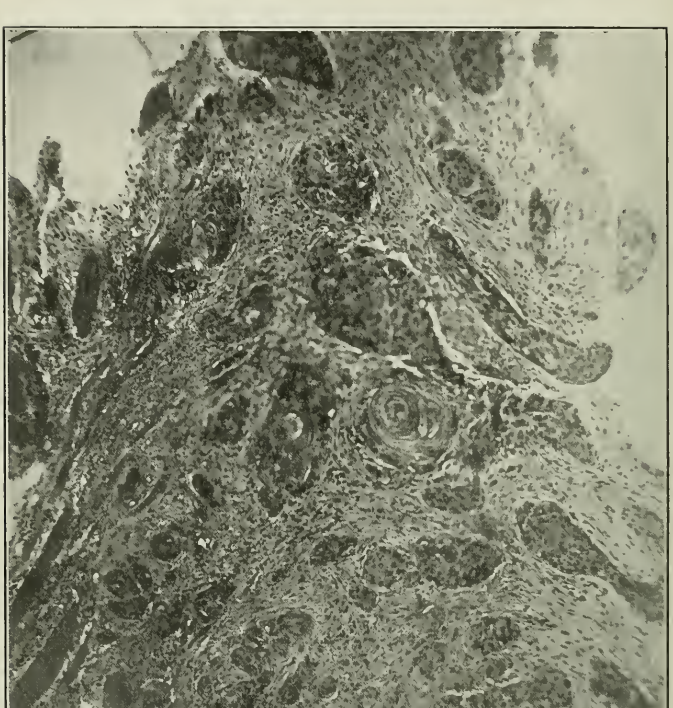

FIGURE 2.

Photomicrograph of the carcinomatous portion. Note the solid nests of epithelial cells of a squamous type.

Pieces of tissue were removed from this mass with biters, for pathological examination.

Report of Doctor Albert on tissue removed from esophagus:

*Gross Pathology:* The material to be examined consisted of tissue scrapings, the mass of which measured about one cm. in diameter, and individual pieces of which measured from two to five mm. in diameter.

*Microscopic Pathology:* Certain pieces of tissue consisted of the normal squamous epithelial lining of the esophagus. Others and more especially the largest piece consisted of connective tissue and non-striated muscle tissue invaded by a large number of solid masses of epithelial cells. These epithelial cells were of the squamous type. Many of the nests or masses of epithelial cells contained hornified epithelium in their centers, forming distinct whorls or pearls. The muscle or connective tissue invaded by the epithelial cell masses is markedly inflamed as shown by a large amount of "round-cell" infiltration; the "round-cells" of which are principally fibroblasts.

Other pieces of tissue contained distinct tubercles consisting of central masses of beginning caseation necrosis surrounded by quite a large number of giant and endothelial cells, and about the periphery, large numbers of "round-cells" consisting of fibroblasts and lymphocytes. The giant-cells were of the typical tubercle type; that is, the nuclei were arranged about the periphery of the cells whereas the center of the cells showed the homogenous structure of caseation necrosis. In one field, fourteen giant cells were found. A few tubercle bacilli were also found.

April 22, 1916. The Wassermann test on the blood was positive.

May 7, 1916. The tuberculin test (von Perquet) was positive.

May 2, 1916. Tubercle bacilli were found in the sputum by Doctor Royce.

May 15, 1916. Smears taken directly from the larynx were reported negative for tubercle bacilli by Doctor Royce.

The blood examination on May 7, 1916, revealed the following:

Hemoglobin 75% Sahli. Red cells 4,900,000.

White cells 12,600.

Differential count: Polys. 71%; Lymphos 21%.

Transitionals 2%; Large mnos. 4%; Eos. 1%.

Basophilis 1%.

The urine was yellow; sp. gr. 1,008; acid; negative for albumin, sugar and sediment. Microscopically a few hyaline casts were found.

The patient was placed on antiluetic treatment April 24, 1916; mercury inunctions and potassium iodide. A marked improvement in dysphagia was immediately noticed.

By May 15, 1916, he was able to swallow all kinds of soft food and semi-solids. He ate meat, potatoes, vegetables, and had no pain or difficulty while swallowing or afterwards.

Microscopical examination of the pieces removed from the esophageal tumor proved both its carcinomatous and tuberculous character. The marked improvement in the swallowing after antiluetic treatment alone, and in the presence of a positive Wassermann, suggests a definite luetic element present in this mixed tumor. Difficulty in swallowing twenty-eight years ago, which lasted six weeks and then disappeared, is also suggestive of syphilis.

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## DISCUSSION.

**Dr. Samuel Iglauer**, Cincinnati, Ohio, referred to the remarkable fact that the esophagus so often escaped tuberculosis, considering the amount of sputum swallowed by the patient. It was probably explained by the fact that the esophagus, like the mouth, owing to its peculiar epithelium, was not predisposed to tuberculosis. Another explanation might be found in the rapidity of deglutition. In investigating this question sometime ago he had found that food passed the larynx in 1-40th of a second. In the absence of abrasions the rapidity of deglutition shortening contact probably prevented infection. On the other hand, tuberculosis of the intestines was very common, probably due to the prolonged stay of the tubercle bacilli in the intestinal tract.

It was very important, after removing impacted food, to look for some pathological lesion. He recalled a case, in a coal heaver, in whom food became impacted in the esophagus, a gumma being found upon removing the food. In another case the peribronchial glands were found to be involved, probably syphilitic.

Several cases of coexistent carcinoma, tuberculosis, and syphilis of the larynx had been reported. Carcinoma frequently supervened upon syphilitic ulcer, but it was very rare for carcinoma to supervene upon tuberculosis of mucous membrane or skin.

He suggested the use of the cautery in the treatment of tuberculous lesions in the esophagus.

## THE USE OF THE ASPIRATION OR VACUUM SUCTION APPARATUS IN CASES WHERE THE ABILITY TO SWALLOW OR COUGH HAS BEEN PARTIALLY OR COMPLETELY LOST AND THE PATIENTS ARE PRACTICALLY BEING DROWNED IN THEIR OWN SECRETIONS.

By ROBERT CUNNINGHAM MYLES, M. D., New York, N. Y.

During the summer of 1914, a patient of the writer's—a man about 67 years old—whom he had treated for a chronic tracheobronchitis, developed bronchopneumonia. He gradually grew worse and for weeks there seemed to be but little for life to cling to; he was emaciated, his heart but feebly fluttered, while his cough did not have the force to eject the thick mucopurulent secretion much beyond the laryngeal region.

A Wappler suction machine was placed beside his bed and used at intervals night and day to remove quantities of tough, tenacious secretion, which clung to the dried oropharngolaryngeal walls. This would give the patient periods of rest and quiet after the violent efforts of coughing and expectorating.

The patient recovered and is well, as far as his respiratory tract is concerned.

The writer does not feel that the use of aspiration was absolutely essential in this case, but believes that it contributed very decided relief at critical times.

The use of some type of suction apparatus, during the past several years—especially in cases of tonsillectomy where the secretions and blood accumulated in the hyoid fossae and laryngeal region—naturally begot the habit of relying on the procedure for relief in all cases where suffocation was threatened.

Two cases came under the writer's care within the past several months, when the employment of suction was one of the essential factors in saving the lives of the patients.

### CASE REPORTS.

Case 1 was in a child, about three years old. I was called to see it as an urgent emergency case. I found the little patient in extremis, with that peculiar noise in the throat that is usually heard in this class of cases just before the end. There was very limited respiratory space, and complete inability to get rid of a flood of thick mucopurulent secretion. All the recesses of the nose, rhinopharynx, mouth and lower pharynx seemed to be filled to capacity with the tenacious mucus.

The cervical lymphnodes were very large. The tonsils and the adjacent parts were extremely edematous, almost in juxtaposition, with the respiratory air churning through the narrow interspace, and the volume of secretion extreme, the child presenting a pitiable picture of emaciation, exhaustion and struggling for air. There had been inability to swallow for several days.

I tried to remove the secretions with gauze swabs. The quantity was so great, the space so narrow, the patient so weak that I discontinued the efforts and having secured a suction apparatus within ten minutes, I rapidly removed an unbelievable quantity of secretion from the mouth, pharynx, nose and the region of the larynx. Some improvement in the respiration was obtained. I forced my finger down the throat and detected a soft mass on the post-pharyngeal wall near the œsophagus.

The child's respiration and heart stopped—apparently he had passed away; but artificial respiration and the reverse position, head downward, gradually restored him.

I passed a long, narrow bistoury through the limited space between the tonsils, and cut—guided by topographical knowledge and limited vision. A free flow of pus followed and it was necessary to hold the child in the reverse position and use the aspiration apparatus to prevent the pus from flowing into the trachea. I inserted the forefinger through the incision, found a cavity about 1½ inches in diameter, and tore the wall freely with my finger.

Convalescence was gradual. When patient had become stronger, his tonsils and adenoids were removed, and he was approximately normal when I saw him last.

Case 2 was in a man aged seventy-two, who had been suffering for a week from an influenzal tracheobronchitis. The writer had treated him for years, at intervals, for attacks of non-inflammatory cough, and also for about an equal number of acute attacks of laryngo-tracheitis.

He consulted me on March 10th. I found the larynx and trachea acutely inflamed, with moderate edema of the mucosa. He was suddenly called on a trip to New England, but returned to my office on the 15th of March in a worse condition, with the symptoms of an extension of the inflammation to the smaller bronchi. I warned him of the dangers of the disease and advised rest and careful attention to the local and general condition.

Messages were sent for me to visit him on the afternoon of the 17th but they did not reach me for several hours. I visited him about 7 P. M. and found that Dr. Thomas J. Tobin had seen him and prescribed sparteine and a cough mixture. His temperature of 102° and a pulse of 98 of poor quality at 4 P. M. had improved—the temperature had dropped to 99.3°. Patient had a fair night, with some severe coughing up to about 4 A. M. when he vomited and became slightly cyanosed, with a weak pulse.

Dr. Tobin and myself arrived about 6 A. M. and found the patient in profound coma—with every reflex and muscle in the body paralyzed except the heart and diaphragm. The respirations were very slow and irregular—two to twelve a minute, Cheyne-Stokes in character. The heart showed almost every weakness of which it is capable.

The mouth, throat, trachea and bronchi were full of thick, mucopurulent secretion, and since he could not swallow, cough nor expectorate, it was evident that he would soon suffocate by drowning in his own secretions.

A Sorensen suction machine was secured between 8 and 9 o'clock. Very indifferent relief had followed the swabbing of the secretion from the pharynx. A string was passed through the tongue, the mouth pried wide open and the tongue drawn well out. It was necessary to use aspiration every ten to thirty minutes. The metal tubes were used in the mouth and pharynx and the rubber ones were passed through the nose into the trachea and bronchi. The relief from the ominous rattling sounds was immediate and magical.

Dr. Stuart Hart came about 9 o'clock. He and Dr. Tobin were most resourceful in making an extremely weak heart continue to beat and a faltering diaphragm to continue to contract.

The eyes manifested no reflex when the conjunctiva was touched.

The larynx was in the cadaveric position and showed no symptom of irritation when the tubes were introduced. The sphincter ani was completely paralyzed. An enema of coffee flowed out immediately without evidence of bowel contraction. No voluntary muscle was seen to move until after 2 P. M.

The first evidence of return of muscle and nerve power was a slight effort to close the mouth at about 2:30 P. M. The mouth had been wide open, without a gag, for more than five hours.

Patient commenced to regain consciousness about 3:30 P. M.; then the reflexes returned and about 7 P. M. he talked rationally.

The vacuum suction apparatus removed the otherwise fatally obstructive secretions from the respiratory tract, and allowed time for making use of nearly every procedure that the science of medicine could supply for stimulating and sustaining the action of the heart and respiratory muscles and for causing the return of the circulation of the blood in the brain.

It would seem to the author that there is a broad field for the use of the suction apparatus in cases of coma, paralysis and inability of self-removal of the secretions from the respiratory tract, from whatever cause.

Five to twelve inches of pressure were found to be the best. The tubes while in use should be kept in a rapid vibratory motion, to prevent the indrawing of the mucosa. The tips of certain models of tubes should have several perforations in order that the air may aid in drawing the secretions through. A variety of terminal tubes is presented—those of the rubber catheter type can be used most effectively through the nose as well as the mouth. I have had an Einhorn duodenal tip placed on the end of a long malleable metal tube, to be used in the lower pharynx and trachea; but would advocate a tip a very little larger than the diameter of the tube for use in the bronchi.

It would be difficult for one to believe that a patient could recover from the condition in which this one was for over eight hours—unless one had been a witness to the experience. It is not quite clear as to what was the exciting or actual cause of the profound temporary paralytic coma. It would seem to simulate the condition brought about by hypertensive action over the celiac axis.

The patient was critically ill for several weeks—with irregular bronchopneumonia symptoms. On April 16th, 17th and 18th he was up in the chair and down stairs on the 18th, and seemed to be convalescing favorably. About 1:30 A. M. on the 20th of April he experienced severe and violent pain in the

right side—from an extension of the inflammatory process of the pleura. On April 27th a section of a rib was removed and the pleura opened and found full of pus.

He failed gradually and expired the next day.

## DISCUSSION.

**Dr. Thomas Hubbard**, Toledo, Ohio, had always been more or less skeptical about bronchial flooding until a recent experience had convinced him of its possibility. He had been called to see a child who had aspirated a grain of corn. The corn completely plugged the right bronchus, where it had been for forty-eight hours. Following extraction the child was asphyxiated by the flood of mucous secretion released by the removal of the foreign body. The patient was held head down, and there was a gradual return of respiration. It was clearly an instance of the sudden flooding following the removal of a foreign body in the bronchus. Such a possibility should always be born in mind in connection with these cases. He should have been forewarned in this case and prepared to aspirate or to give oxygen, as the physical signs indicated complete plugging of one bronchus, the right. The absence of any respiratory sounds and dullness of the right side, without fever, pointed clearly to pent up bronchial secretions. Dr. Myles' method should be available for such cases.

**Dr. Harris P. Mosher**, Boston, Mass., said Dr. Jackson had brought before one of the societies, and had reinforced in his book the great advantage of direct bronchoscopy in the relief of bronchial flooding. In the second case cited by Dr. Myles, which was a typical case of retropharyngeal abscess, incision would have accomplished all that suction did. He felt that the vacuum suction apparatus would be of great value in cases of pneumonia in which the patients were drowning in their own secretions.

**Dr. George F. Keiper**, Lafayette, Ind., called attention to the use of the suction apparatus for the removal of secretions from the mouth in cases of children with stricture of the esophagus due to swallowed lye. Children, especially very young children, when the esophagus was dilated, developed enormous amounts of secretion under these circumstances, until they would become blue in the face, and it would be necessary to withdraw the dilator from the esophagus until the secretions were removed. With the use of the suction apparatus for withdrawing secretions from the mouth and trachea he had found it possible to dilate these strictures for a longer time than would otherwise be possible. He had used Dr. Beck's' apparatus. Several years ago he had reported before this Society the treatment of severe cases of asthma by the introduction of the bronchoscope and the use of adrenalin-cocain solution applied locally to the bronchi. The secretion could be withdrawn by suction after the introduction of the adrenalin-cocain solution.

**Dr. Joseph C. Beck**, Chicago, Ill., cited a case of very severe bronchorrhea, of several years' duration, in a woman who had been at an institution for the treatment of tuberculosis. The patient was unable to expectorate the large amount of secretion. He put her under suspension laryngoscopy, and every day for three weeks withdrew quantities of this secretion, with the result that she recovered, and regained thirty or forty pounds of weight. This was not tuberculosis, but merely bronchorrhea, probably secondary to nasal infection.

**Dr. William H. Haskin**, New York City, recalled the fact that in 1909 or 1910 he had produced and had presented the vacuum appar-

atus and method of treatment, which was received as a joke at the time. He had repeatedly emphasized the usefulness of the method in many of these conditions. In cases of retropharyngeal abscess it has been his custom to attach a trocar to the vacuum cleaner, and remove the secretion, obviating the danger of any secretion getting into the trachea. It was very gratifying to see that the instrument makers were hardly able to supply the demand for the pumps since their value had become so fully recognized.

# THE OCULAR CIRCULATION AS AFFECTED BY THE DISEASES OF THE NASAL ACCESSORY SINUSES.

By LEWIS A. COFFIN, M. D., New York, N. Y.

During the last decade much interest has centered about the question of the dependence of certain diseases of the eye, the optic nerve and the neighboring tissues upon diseases of the nasal accessory sinuses, especially empyemea of those cavities.

In a paper read before the New York State Society in 1912, in discussing latent sinusitis[1] I said: "Such a disease may exist in the sinuses of persons whose noses present a practically normal appearance. It may involve any of the sinuses, and is of the utmost interest and importance because of the difficulty of diagnosis. When involving the frontal or antral cavities transillumination and the radiograph materially aid in the diagnosis, but existing independently in the ethmoids or sphenoids or confined to those two cavities, the diagnosis must rest upon symptoms and possibly the study of the eye." At that time Dr. Frank Parker, an oculist, was associated with me and all suspected cases from either the eye or sinus side of the case were studied together. I was struck by the number of cases in which he reported from an examination of the fundus that the veins seemed larger and tortuous, while the arteries seemed somewhat small as compared with the normal, the difference frequently appearing in the different sides of the same patient. We arrived in a general way at the conclusion that the most frequent cause of eye disturbances arising from accessory sinus disease was due to circulatory changes.

The intimate relationship as to proximity of the optic nerve to the various sinuses, especially the posterior ethmoids and the sphenoids has been shown by the painstaking work of Onodi and Loeb and this showing has always been accompanied by the assumption, if not the direct statement, that on account of this relationship the nerve is frequently affected by the direct influence of the toxins of the sinus pus or organisms.

Disease of the eye and its nerve dependent upon the diseased accessory sinuses, is I think, universally admitted at this time, but a considerable diversity of opinion exists as to the explanation even in cases almost identical as to symptoms, signs, etc. J. Herbert Parsons, authority on "Pathology of the Eye" in discussing certain

symptomatic diseases of the eye, says "The similarity of the histological appearances depicted by various authors is striking and the chief differences are to be found in their interpretation." Again having discussed angiosclerosis, stooping, tortuosity of the veins and conditions on the arterial side of the capillaries as possible elements in causing thrombosis of the central and other orbital veins, he says "when all the above causes have had due weight there still remains certain cases not easily explained. These are cases occurring in young people without cardiac or renal disease." That a better understanding of the matter may be had from a study of the circulation of the nose, as well as that of the eye, is my apology for this present effort. The circulation of the nose and eye are most intimately associated, a large part of the blood supply to the nose being brought to that cavity by the ethmoidal branches of the ophthalmic artery and returned by the ethmoidal veins to the ophthalmic veins.

This particular arrangement lends itself to what to the writer's mind is the most frequent affections of the optic nerve and eye, viz., circulatory changes brought about largely by the inflammation of the veins due to the direct continuity of pyogenic tissue.

As many of you know this view has been held by the writer for some years and he confesses that it has some times irritated him that he has not been able to get his friends, especially the oculists, to see as an explanation of many eye affections, circulatory changes due to sinus disease.

Dr. Frank Crane of the New York Globe says we are all governed more by the ghosts of former beliefs, dicta, etc., than by any array of concurrent facts and logic that can be made.

Let me introduce some of the ghosts which, it seems to me, have dominated our thought in this connection.

The first is that of the assumption before alluded to that has gone forth with the anatomical work showing the intimate neighborhood relationship of certain nasal accessory sinuses and the optic nerve —that the nerve is directly poisoned by the toxins from the sinuses.

The second arises from beliefs established by the reading of such statements as the following from Parson's book on Pathology of the Eye[2]. "Orbital cellulitis occurring in young children seldom arises from the accessory sinuses of the nose, since these are relatively undeveloped up to eight years of age."

Another is perhaps the most formidable of all and arises from the teaching of years that certain eye affections are but the local

expression of constitutional vice or cachexia. Things already explained need no further explanation. There are others that I shall not mention, but I sometimes feel that if the simple naked truth be introduced to any of us who have seen and harbored any of these ghosts, truth, at times, gets scarce an inclination of the head in recognition to say nothing of the extended hand of welcome. Notwithstanding, however, I am going to introduce some young truths that have not as yet been entirely taken up by society.

First let me quote from Dr. Keen's Surgery[3] a part of his article on phlebitis.

After classifying phlebitis as aseptic, toxic and septic, he again sub-divides septic phlebitis into, first, non-pyogenic phlebitis, and, second, pyogenic or purulent phlebitis.

"Septic non-pyogenic phlebitis develops in the course of infectious diseases as an intercurrent infection, also resolves in a benign way after going through a non-purulent course. Changes in the inner coat are of toxic origin rather than of direct bacterial etiology. But as the toxemia is dependent upon bacterial activity (toxins, tox-albumens, etc.) and upon the presence of pathogenic (non-pyogenic) cocci which may have colonized in some parts of the organization outside the seat of the phlebitis, we should group these as septic, non-pyogenic phlebitis. This group includes probably the majority of the phlebites which occur in the general fibrile infections; typhoid, typhus, pneumonia, the exanthemata, influenza, appendicitis and postoperative states.

Septic pyogenic or purulent phlebitis is the second and more formidable division of the septic group. It is essentially dependent for its causation upon the localization and colonization of pyogenic microorganisms (staphylococci and streptococci) in the venous walls, thus accounting for the great tendency to migration and metastasis displayed by infections of this type. The most notable examples are the progressive and purulent phlebitis of the lateral or sigmoid sinus consequent upon middle ear diseases, the especially malignant type of facial thrombophlebitis caused by carbuncles and furuncles of the upper lip, the disease spreading rapidly through the ophthalmic vein to the cavernous sinus, thence to the meninges and the brain and the pylophlebitis or portal phlebitis after intestinal, peritoneal and especially appendicular infections.* In all these forms of phlebitis (excluding the aseptic) progressive purulent thrombosis occurs in the veins. Hence the term thrombo-

*He might have mentioned also the phlebitis of the ophthalmic veins as due to the empyemic disease of the nasal accessory sinuses.

phlebitis which is now synonymous with phlebitis because of the universal association of blood clotting with the infections lesions of these vessels.

The prefixes peri-, meso- and endo- are applied as in arterial pathology, to distinguish the starting point or seat of most marked lesions, all forms of phlebitis invariably involve all the tunics and reach the endothelium or intima either as an initial or terminal process.

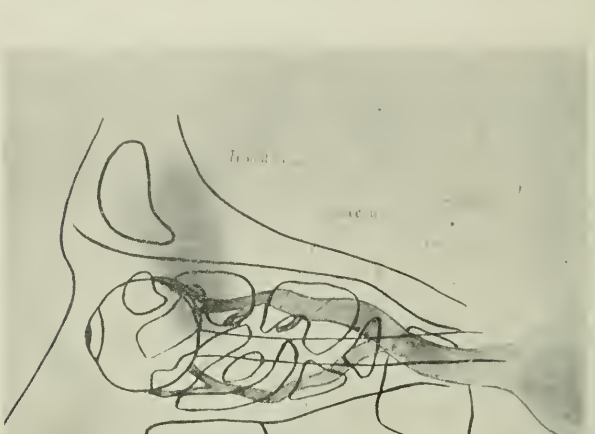

Fig. 1. Diagram showing relations of ethmoidal and ocular circulation.

Phlebitis may be acute, sub-acute or chronic, partial circumscribed or diffuse.

Etiology and pathogenesis. The toxic or septic form is associated with the pyogenic organisms staphylococci, streptococci, pneumococci, etc.

Phlebitis spreads most frequently by direct continuity or surface, as in the malignant thrombophlebitis of the facial veins following furuncles or carbuncles of the upper lip."

This picture (Figure 1) is a diagram in which I have endeavored to show some of the relations of the nasal and orbital circulation. The cloudy lines with dark edges represent the ocular and ethmoidal veins, the enlargement on the right representing the cavernous sinus. The spaces outlined by the dark lines represent the accessory sinuses of the nose, and the space between the parallel dark lines, the optic nerve.

Where exist then such an ideal condition for the production of a phlebitis? To appreciate all the pathological changes that may take place in the eye from this relationship, one must understand the various phases of phlebitis. And having arrived at such an understanding or near it he should think of the various veins of the orbit and bulb; then picture to himself the various effects of these various phases of phlebitis on the various veins and he will almost think that the list of diseases of the eye is not sufficiently long.

Direct poisoning of the optic nerve by toxins from the diseased accessory sinuses may take place, but if occurring at all, it can account for but one form of disease.

DeSchweinitz[4] in the 1916 edition of his book, Diseases of the Eye, says "Orbital cellulitis may be caused by the extension of inflammation from diseased teeth or suppuration in the ethmoid cells, sphenoid, or antrum of Highmore—Birch-Hirschfeld's investigation demonstrates that the largest number of orbital inflammations (about 60 per cent.) are due to accessory sinus inflammation. The infection is conveyed from the sinus to the orbital contents by means of septic thrombosis, thrombophlebitis, lymphangitis, or erosion of the bony partition. In pyemic cases, and indeed in the course of any severe inflammation of the cellular fatty tissue of the orbit, *phlebitis* of the *orbital veins* may become a complication and extend to the cavernous sinus, leading to a fatal termination.

Preysing[5] in 1898 reported a case of orbital cellulitis in a child five years of age as having empyema of the ethmoids. No autopsy, diagnosis is on anatomical grounds.

Vossius[6] in 1900 in discussing the affections of the orbit from inflammatory disease of the accessory sinuses emphasizes the probability of the suppuration in the orbit arising from a primary thrombophlebitis of the ethmoidal veins extending to the superior ophthalmic vein, etc., and reports a case. Among others, who have discussed orbital phlegmon and ascribed the cause to thrombophlebitis having its origin in the diseased accessory sinus are Gutmann,[7] Zarniko,[8]

Hajek,[9] Kuhnt,[10] Logan Turner,[11] Denis and Vacher,[12] Clermont[13] Bruckner, [14] Wurfel, [15] Courtax,[16] Birch Hirschfeld,[17] Ziem,[18] Weiner[19] and Ball,[20] in discussing relations between eye and nasal disease mentions several cases of iritis which did not yield to original or customary methods of treatment until the co-existing nasal suppuration was treated, and Kuhnt indorses this view.

Ziem arguing that on account of the numerous anastomoses which exist between the vascular supply of the nasal cavity and its accessory sinuses on the one hand and that of the orbit on the other, says a hyperemia in the first named territory must become propagated to the other or orbit, to the interior of the bulb and especially to the ciliary body, etc.

A short time since wishing to get a series of X-ray findings as to the accessory sinus of children in health and diseases, I was for a time troubled to find the diseased sinuses until I thought of the children's Eye Ward of our hospital. I asked Dr. Law to X-ray some of the heads from that ward. They were chosen as to age and every one showed diseased sinuses, furnishing strong presumptive evidence of the inter-dependence of the diseases of the eye and the nasal accessory sinuses.

## CASE REPORTS.

*Case 1.*

W. R., age 1½ years.

*History.* March 1st. Complaint: Protusion of left eye.

*Family History.* Negative.

*Personal History.* Has had discharging right ear for past few months. Also has had convulsions—not definitely characteristic. No past sickness, though never has been a robust child.

*Present illness.* Two weeks ago left cheek became suddenly red and swollen. In one week this spread to the lower eyelid. About four days ago the eye started to become prominent and upper lid swollen. At no time was any nasal discharge seen.

*Physical Condition.* "General pallor—dorsum of feet edematous." Otherwise negative except local condition.

Left eye. Marked exophthalmos. Eye seems pushed down and out. Chemosis upper bulbar conjunctiva. Cornea ulcerated. Scar over brow of incision previous to admission.

Right ear. Thick purulent discharge through a small central perforation in the drum which is bulging and lacerated.

Temperature 98.6°. X-ray sinuses cloudy.

Leucocyte count 22,000—polynuclear neutrophiles 78%.

*Operation.* External canthotomy.

(Dr. Hume.) Incision of skin above globe and no pus found in orbit. Anterior ethmoids opened and found clean. Wound closed.

Postoperative course. For next week the baby seemed to improve. The eye was less prominent. Ethmoid wound healed nicely. Temperature fluctuated from 99° to 104° and on last day was normal. Corneal ulcer increased in size.

Second operation by Dr. Coffin.

Radical opening of posterior ethmoids, sphenoid and antrum. No free pus found.

During the week following this the baby got steadily worse. Wound broke down and considerable pus. Temperature 100°-102°. Blood count 20 to 42,000. Eye ruptured. Right foot became swollen and was incised—much pus.

At the end of this week—March 15th—the child died.

Tissue from the regions of the various sinuses were submitted to Dr. Dwyer, pathologist, to the hospital who reported the following findings:

Patient—Lillian Reimer—No. 853,460, Lab. No. 6,373.

*Tissue report.*

From antrum—Tissue swollen and edematous. No round celled infiltration with the exception of the immediate areas around the blood vessels.

From ethmoids—(anterior). Marked edema of the tissues. Round celled infiltration around the blood vessels and around the glandular tissue. Here and there masses of pus cells and leucocytic infiltration.

From posterior ethmoids. Here the most marked pathological changes are shown—marked round celled infiltration; large numbers of pus cells with here and there broken down tissue: hemmorhagic areas marked. Here and there there is thrombosis of the smaller blood vessels.

(Signed) J. G. Dwyer, Pathologist.

These were the autopsy findings as to head, the only part autopsied. A small opening was found in cribriform. Brain and coverings normal. Cavernous sinuses clear. A block of bone containing sphenoid and ethmoid sinus together with optic nerves and posterior half of diseased eye were taken for pathological study. This specimen was referred to Dr. John E. Welch, who reported as follows:

*The ethmoid.* The tissues lining the ethmoid cells show an inflammatory process. The fibrous portion of this structure is infiltrated by serous fluid of the inflammatory process and the constituent fibres are widely separated by this inflammatory product. There is an extensive exudate of mononuclear and polymorphonuclear leucocytes which extends throughout the tissues. The polymorphonuclear cells of this exudate predominate and are found in much greater numbers permeating a zone adjacent to the epithelial covering and to a much less extent in the tissues lying immediately over the bone.

The walls of the blood vessels show cloudy swelling and near the bone the veins show extensive thrombosis. These thrombi are both parietal and occluding and show numerous leucocytes enmeshed in fibrin. The thrombosis is not confined to the vessels in the tissues just over the bone. Some thrombotic veins are also found in the outer zone of the tissue near the epithelial covering but in this region they are much fewer in number than in the area near the bone.

*Orbit.* The tissues of the orbit show a general inflammatory condition which involves the fibrous and areolar tissue extensively and to a less extent the muscles. The fibrous tissue septa are congested and infiltrated with leucocytes mostly of the polymorphonuclear variety. The blood vessels show cloudy swelling and about some of the vessels there is an extensive exudate

or polymorphonuclear leucocytes in the adventitia. The bands of fibrous tissue that penetrate the ocular muscles also show an exudate of polynuclear leucocytes.

The veins show thrombi which, in one instance, in the case of one large vein, the thrombus is purulent. The muscle fibres of the ocular muscles show cloudy swelling.

*Optic nerve.* Cross section of the optic nerve just behind the eye ball shows a thrombus in the central vein which occupied about fifty per cent. of the lumen of the vessel. The thrombus is attached to about one-third of the circumference of the intima of the vein. The clot is composed of fibrin in the meshes of which are a few leucocytes.

*Case 2.* This case is of interest because of the age of the patient, and also from the fact that a diagnosis of accessory sinus disease could be more readily made from a study of the eye than through any other clinical symptom.

He was a man 78 years of age, whose case progressing unfavorably especially as to the chemosis and the cornea, I was asked to see. I made a diagnosis of pansinusitis of the left side. The following day I did a radical Killian. Dr. Welch stood by and took specimens from the various regions operated. He reports them.

*Frontal sinus.* Sections through the lining removed from the frontal sinus show the epithelium of this membrane intact and apparently in a condition of hypersecretion. The supporting fibrous tissue is the seat of a chronic inflammation which has produced considerable thickening in places. The blood vessels are very few in the most thickened parts but in others they appear to be increased in number. Some of the larger arteries have thickened walls. Through the membrane in certain areas there are leucocytes among which are numerous eosinophyle cells.

*Ethmoid.* Sections through the tissue scraped from the ethmoid cells show a general inflammatory condition. The fibrous tissue stroma is thickened by production of fibrous tissue and acute inflammatory products. The vessels are congested and their walls show cloudy swelling. They contain, in almost every instance, a great excess of leucocytes and some appear to contain leucocytes exclusively. Some of the veins show thrombi, especially some of the larger ones, a few of which have cavernous dimensions. The epithelium covering the surface is intact but shows cloudy swelling.

*Tissue from the orbit.* Sections through material taken from the orbit show necrotic fibrous tissue which is infiltrated by an inflammatory exudate or leucocytes and serum. Some of the small veins near the periphery of this tissue show hyaline thrombi in which the constituents of the thrombus are fused together.

The old gentleman is up around feeling well. Swelling of orbital tissues much reduced as is the chemosis and the cornea is apparently clearing up; an unlooked for benefit.

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## DISCUSSION.

**Dr. Edgar S. Thomson,** New-York City, speaking from the ophthalmological point of view, said: The influence of the sinuses upon the eye and orbital tissues occur in two main groups; intraocular, and orbital inflammations. Orbital inflammation, or orbital cellulitis, in the vast majority of cases, is due to sinus infection, unless, of course, the infection comes through an external wound. In fact, when we consider the external protection afforded by the facial walls, it is difficult to explain idiopathic orbital cellulitis on other grounds. Infection from the antrum or frontal sinus manifests itself so far forward, and the symptoms are so characteristic, that it is not necessary to do more than mention them. The most common cause of orbital cellulitis is direct infection from the ethmoids. It is important to recognize this in its early stages. The symptoms are slight exophthalmos, pain on rotating the eye, and the usual accompaniment of slight rise in temperature.

The best means of determining the exophthalmos is through the exophthalmometer of Hertl, which is, no doubt, familiar to you all. The reading of this instrument can be considered from its absolute figures, unless one eye shows a very much higher reading than the other, without a marked asymmetry of the face to account for it, but the comparative reading showing a gradual protrusion from day to day.

Inflammation of the ethmoid also frequently causes a low grade type of choroiditis, in the course of which numerous fine opacities are thrown out of the vitreous. I have repeatedly seen good results from opening the ethmoids in such cases.

Inflammation of the sphenoid most frequently causes inflammation of the optic nerve. This occurs at times when no other symptoms of sphenoid disease are present. The neuritis is usually inflammatory in type, and is well advanced on one side while the other optic nerve is normal. It is frequently necessary to explore the sphenoid in such cases without other symptoms being present. Nor is it wise to wait too long on account of the danger of atrophy of the optic nerve.

It is very important for the sinuses and the eyes to be studied together. The eyes should be examined in case of sinus disease, and the sinuses should be carefully examined and perhaps explored if the type of ocular inflammation is suggestive.

**Dr. Christian R. Holmes,** Cincinnati, Ohio, was interested to hear a paper on this subject. In a paper read before the Society ten years ago, at Pittsburgh, on the relation of the sinuses to the orbit, he had made the statement that forty per cent. of all affections of the eye, including asthenopic conditions, could be attributed to some form of sinus disease. As a young physician, twenty-five years ago, he had succeeded in making an impression upon a certain rich clientele by holding that affections of the eye could be treated successfully by attention to the nose and sinuses. Two children of a wealthy family had been cured, as if by magic, and the family had been good patients ever since. In 1896 he had reported the first case, so far as he knew, of the sphenoid being responsible for atrophy of the optic nerve. He presented plates at that time. The case was one of one-sided atrophic change. The man suffered intense pain,and had become almost totally blind in the affected eye, so that it became necessary to act promptly. After the general surgeon, the neurologist, and the ophthalmologist had failed to relieve him, the speaker decided to open the sphenoid, and succeeded in giving relief of all pain and gradual restoration of almost perfect vision. This patient had been under observation at intervals up to six months ago, when he died from pneumonia.

Ten or fifteen years ago, when rhinologists were using the cautery rather freely, he had seen retinal hemorrhage result from the too free use of this instrument in the region of the middle turbinated bodies. He had also seen extensive thrombosis of the retinal vessels from a burn by the cautery. One of the most remarkable cases he had witnessed was that of a doctor, who was blind in the right eye, and growing so in the left eye. X-ray examination revealed pansinusitis. The man had iritis, floating masses in the vitreous, and detachment of the retina. He performed a Killian operation (radical). The iritis cleared up, the exudate was slowly absorbed, and the detached retina became reattached. The man is still practicing medicine.

**Dr. Hanau W. Loeb,** St. Louis, Mo., considered anything important which added to the understanding of the vascular relations between the nose and the eye; Dr. Coffin's paper, therefore, was a distinct contribution. The facilities which Dr. Coffin possessed for studying eye cases rendered his observations of value to this Society and others to which he belonged. The speaker's own work had been to show anatomic relations, but in no sense would he deny that the diseases under discussion could not invade the orbit through the veins; they did not jump from the nose to the eye. They must pass through some medium. By means of autopsies and pathological studies of cases of this sort it would be possible, in time, to reach more definite conclusions than were now warranted. He had had three cases of blindness due to ethmoidal disease, all of which were cured. The most characteristic of these was a boy, sent to him on account of inability to see more than the fingers immediately in front of his eyes. Immediately after cocainization without any other treatment whatever the patient saw a man walk across the street. He wished especially to emphasize that ethmoidal disease was extremely common, whereas sphenoidal disease was less common. The relation of the sphenoid to the optic nerve was common; of the ethmoid less so. He wished again to present the thesis that with the nerve running along the posterior wall of the last ethmoidal cell involvement of the optic nerve would more easily follow in ethmoidal disease.

**Dr. John E. Brown,** Columbus, Ohio, called attention to the possibility of the subsidence of an acute infection of one or more of the

accessory sinuses and the subsequent development of ocular symptoms that would lead the patient to consult an ophthalmologist. At this time careful intranasal examination, and in rare cases, even X-ray findings, might be negative. He had seen such cases. He had also seen others in which a history such as the above was not clear, but in which the usual treatment directed against the ocular—or optic nerve —lesion was not effectual, but when the ethmoid cells had been thoroughly drained by operation, there was a prompt disappearance or relief of the eye lesion.

**Dr. Joseph A. White,** Richmond, Va., referring to Dr. Thomson's statement that it was hardly necessary to take the maxillary sinus into consideration, cited a case. A lady had consulted him in July, 1916, for eye symptoms, headache, etc., but with vision of 20/15. She was relieved by the correction of a low grade astigmatism. In December she returned, with vision of 20/70, headache, and eye pain. X-ray examination revealed diseased teeth, with an abscess at the root of one tooth. The tooth was removed and the vision returned to 20/15. On February 4, the vision again became bad. The X-ray examination had shown no antral trouble, and transillumination was unsatisfactory. An exploratory opening was made in the antrum and pus found. It was irrigated, with immediate restoration of vision. On February 27, the vision was once more affected, and was restored by irrigation of the antrum. On March 24, she came again with bad vision. The ophthalmoscope showed nothing abnormal. The antrum was washed out and shreds of mucus found, but no pus. Vision was at once restored. This case, left alone, would probably have progressed to optic atrophy. Each time the vision failed irrigation of the antrum restored the sight to normal. The case illustrated the relationship between the optic nerve and the maxillary antrum. As the case was still under treatment a further report of its progress would be made.

**Dr. Coffin,** in closing the discussion, expressed the hope that a more sympathetic relationship with ophthalmologists would enable him to continue his investigations. Disease of the antrum, the sphenoid, the ethmoid, even disease of the nasal cavities, came in the same category with reference to the relationship to the optic nerve. Involvement of the cell most distant from the nerve might, through the circulatory relationship, give rise to trouble.

# AID FOR THE DEAF.

By CLARENCE JOHN BLAKE, M. D., Boston, Mass.

The purpose of this address is to urge the differentiation and consideration of those cases of slowly progressive middle ear disease with, ultimately, extreme impairment of hearing, which are only moderately amenable to active treatment but are, however, amenable to retardative measures consisting of simple self applicable treatment, long continued, under occasional competent professional observation and advice including, in addition to such local treatment as may be demanded at the time, suggestions, either directly or through the general physician, as to general hygiene, compensations for fatigue incident to grave impairment of hearing, or the treatment of other conditions of the body bearing upon the aural disease.

Among the cases of long continued impairment of hearing which challenge the desire for amelioration, however moderate, are those in people who, accustomed to fair participation in the audible communication which makes an important part in daily life, have become aware of a gradually increasing expenditure of compensatory effort in the achievement of that which had previously been an unconscious possession, an illustration of the fact that people with normal hearing have at their command nearly double the amount of this sense function than that absolutely required for the customary demands of social converse and that, therefore, very nearly half of the hearing power, in this respect, may become progressively lost without individual cognizance of the change.

Disruptions of the sound transmitting apparatus of the middle ear or affections of the labyrinth with concomitant symptoms and much impairment of hearing present a distinct contrast to the cases of slow progress in the impairment of hearing amenable to retardative treatment of a simple nature, continuously employed.

As contrasted with cases in which a solution of continuity of the sound transmitting apparatus, either the result of trauma or of pathological changes, is the cause of an impairment of hearing to which other forms of repair effort are applicable, in the cases immediately under consideration the sound transmitting

structure remains integrally intact and the major, and primary, changes are those which concern the two extremes of the acoustic balance, the membrana tympani and the tensor tympani muscle, the minor, secondary, changes being those which occur in the medium of communication with the labyrinth, the articulations of the ossicular chain.

The variety of these changes is considerable and the resultant complex liable to be diagnostically confusing but for the fact that the majority of them present unmistakable evidence of the inceptive cause and fall immediately into the category of tension anomalies which, taken with the case history, gives the key to the situation.

The great majority of the cases to which the continuous treatment is especially applicable belong under a classification which has gradually become defined, under more accurate pathological understanding, as chronic catarrhal inflammation of the middle ear with the nose, or nasopharynx as the fons et origo mali, acceleration of the general middle ear affection being influenced by the existing and varying conditions in these contiguous parts

Of earliest manifestation among the tension changes in the sound transmitting apparatus are those incident to rarefaction of the intratympanic atmosphere, as a result of closure of the tympanopharyngeal tube, this condition involving, as its sequence, increase of circulation in the tympanic mucosa together with tissue changes of a more or less permanent character, including a change in the contour of the membrana tympani, through preponderating atmospheric pressure from without, a relaxation of the tensor tympani with a resultant overcontraction of that constantly operative muscle, a change in the major articulation of the ossicular chain which, if the contraction of the tensor tympani is sufficient, may involve also the minor articulation, between the incus and stapes, by pressure of the inferior articulating facet of the malleus downward upon the incus and thus the institution of interstitial changes in the articulations themselves. Left to itself, without surgical interference, if the closure of the tube is of short duration, the normal conditions of air supply to the tympanic cavity are restored, the congestion of the tympanic mucosa decreases, and the normal atmospheric pressure upon the inner surface of the membrana tympani is restored.

With more protracted, or frequently repeated, tubal closure, the sound transmitting apparatus of the middle ear undergoes changes which, while they may not be microscopically evident,

minutely disturb the function of the acoustic balance, for, even if no structural change has ensued in the membrana tympani itself, the tensor tympani has acquired a new position in its relation to the resiliency of the membrana tympani, which further tubal closure will increase and which surgical interference will aggravate when expressed in the form of too vigorous inflation of the middle ear, an excess of atmospheric pressure in that cavity, under these conditions, tending, not to overcome the retraction of the muscle, but to weaken the resistance of the membrana tympani, especially in its posterior superior segment, and thereby favor a still further compensatory muscular contraction and, finally, that rotation of the malleus upon its long axis, which still further impairs the mobile function of the major ossicular articulation and prejudices the incudostapedal articulation by forcing the incus, and with it the stapes, downward, thus favoring ultimate stapedal fixation also. Taken in the sequence of events the changes incident to the slowly progressive inhibition of activity of the sound transmitting apparatus of the middle ear are, following the tubal closure and rarefaction of the intratympanic atmosphere with the consequent depression of the drum head, cumulative contraction of the tensor tympani muscle, not of its tendon which remains a fair constant in its integral structure, but of the constituent bundles of the muscular tissue itself with resultant lateral traction upon the malleus, turning the short process of the malleus forward, as well as projecting it downward, taking up the slack in the pars flaccida and in the posterior folds of attachment of the short process and putting an additional strain upon the posterior segment of the membrana vibrans, this constituting the secondary stage of the technical effect of the tensor tympani contraction, which is to be regarded as an uncounteracted exhibition of the normal function of a peculiar muscle the recognized purpose of which is the maintenance of a constant strain upon an inelastic membrane, the overcontraction of the muscle being not the result of a primary exaggerated effort, but the sequence of the relaxation of the counterbalancing resistance. With restoration of the intratympanic atmospheric pressure, either spontaneously or by artificial means the normal attitude of the component parts of the acoustic balance is not restored, the resilient membrane does not entirely resume its tension and the muscle does not relax its effort, there is still some stretching of the membrane, less evident than it otherwise

would be because of the overcontraction of the muscle, a stretching most pronounced in the posterior and posterior superior segments and evidenced plainly, in its later stages, by even moderate autoinflation of the middle ear. In the earlier stages of the tensor tympani contraction the hearing for tones of medium high pitch, the differential overtones of the consonant sounds for instance is interfered with because the undue fixation of the sound transmitting apparatus impairs the response in vibration to tones of short wave length, later, with the establishment of a flaccid posterior segment the hearing for tones for medium low pitch, speech fundamentals, becomes impaired; it is before this stretching has occured, while the membrana vibrans is still, as a whole, resistant, that its tension may be utilized toward overcoming the pull of the tensor tympani, this being effected by slow, long continued exhaustion of air in the external canal, and the creation of a partial vacuum with resultant preponderating atmospheric pressure in the middle ear and a gentle, but protracted, push against the muscular contraction.

Closing the external canal at the meatus by means of a conical plug of absorbent cotton and simple cerate, with a thread protruding from its base, firmly anchored in its centre, for purpose of removal of the plug, results, through normal absorption of air by the skin lining the canal, in the creation of a partial vacuum which may be maintained so long as the plug remains in place; the original manometric measurements of Politzer suggested the feasibility of the establishment of a partial vacuum in this manner and subsequent clinical results have demonstrated its usefulness. The plug should be preferably inserted at night, when there is to be no movement in talking or in chewing which would move the external canal and break the air tight seal and it should be removed in the morning, or earlier if it causes undue discomfort, and it may advisedly be applied several nights, as a part of the initial examination of a case of this kind, to determine the degree in which the tensor tympani contraction is a factor in the immobilization of the sound transmitting apparatus and thus facilitate a prognosis as to the degree of improvement in hearing to be effected by helping toward an estimate of the other factors in fixation.

So long as the membrana vibrans remains firm it may be utilized as a medium toward the correction of such obstacles to sound transmission as may pertain in hindrances to movement

of the members of the ossicular chain, more especially in their articulations, and this can be effected by means of the auto-massage of Hommell of Zurich for which we are indebted to his experiments upon himself in his effort to achieve some form of continuous retardative treatment for his own progressive impairment of hearing unrelieved by the occasional ministrations of his colleagues.

This consists of varying degrees of movement of the tragus inward and outward by means of the finger tip, the muscular and ligamentous attachment of this individual plate of cartilage serving for its automatic return after pressure inward, actuating the column of air in the canal, increasing the superficial circulation and gently exercising the drum-head. Firm and sustained pressure upon the tragus with a sudden release, a form of successive massage, which may be practiced from a dozen to a hundred times several times daily, up to the limit of external discomfort or deep-seated pain, serves to favor reposition of the fibres of a stretched portion of the membrana propria and to mobilize the articulations of the ossicular chain, as is demonstrable by the effect observable in the normal ear, with a transparent membrana tympani, by the resultant stimulation of the local circulation. The massage which includes the whole auricle, actuated by the palm of the hand, is too violent, in the majority of cases, and in the end is less effective for purposes of gradual and more permanent mobilization, and therefore may be placed in the same category with the mechanical massage apparatus of Siegle and Delstanche, the use of which, except for diagnostic purposes, is inadvisable and for routine treatment indefensible, since the exaggerated air pressure, in proportion to the tensile strength of the drum-head, together with forcible middle-ear inflation, are to blame for many cases of stretching of the posterior segment.[1]

When the drum-head has been irregularly thickened by previous disease or if there is a conglutinate perforation, the tensile strength of the drum-head, for massage purposes, is merely that of its least resistant portion, and some form of reinforcement may be required, either the familiar paper splints or, for very limited areas and for surfaces beyond the limit of the membrana vibrans, contractile collodion, it being remembered that an excessive or too frequently repeated use, by constricting the superficial

(1) Tension anomalies. C. J. Blake, Archives of Otology, 1902.

circulation of the surface to which it is applied may institute a necrotic process of, ultimately, considerable extent even to destruction of the entire posterior segment.

The efficacy of these simple measures, the auto-massage and the application of the Politzer plugs together with systematic gargling, for its mechanical effect, and, in some instances also faucial finger tip auto-massage, has been sufficiently demonstrated in practice in the past thirty years to warrant their consideration as either a supplementary, or a basal, part of the treatment in many cases of middle-ear fixation.

In addition to its material value in these protracted cases, a continued treatment which is largely in the hands of its subject stimulates interest and effort and helps to ensure that interdependant relationship which should exist between the aurist and his chronic patient, enhancing the responsibility of the latter and affording the former justifiable opportunities for observation and advice.

In the face of the demand for service in acute trouble the minor remedial possibilities may be easily overlooked, but it is well to consider that a gain in the hearing, in a case of chronic impairment, while an insignificant proportion of the normal faculty may be a large percentage of that which the patient possesses. In the study of the needs of that portion of the community which is the subject of this form of an only moderately remediable infirmity, but for which there are attainable ameliorations and compensations, there is a large field for humanitarian effort in which the aurist, as a part of his sociological function, should actively participate.

The patient who can be only partially relieved by physical means and whose handicaps must be carried through life, should be helped to carry it, to understand his limitations, to foresee, as far as possible, his hearing future and to ameliorate his condition upon the basis of his continued and active association with the hearing people about him effecting this by means of some artificial aid if necessary or by recourse to the study of some form of speech reading and preferably that which trains not only symbolically, in the fixed facial positions emblematic of the consonant checks, but in the transitional movements also.

The use of artificial aids to hearing became much extended soon after the invention of the telephone with the substitution of some form of "telephone set" apparatus for the cumbersome and unsightly sound collector. Of these instruments there is now a

great variety, all on the same principle and consisting of a telephone, some form of microphone and a portable dry battery. The telephone is, virtually, the same telephone as that originally constructed but the microphones vary mechanically and are gradually being made to respond more to the range of the voice tones and less to the disturbing metallic overtones which are a customary part of our acoustic environment, but none has as yet been achieved which is an entirely discriminating instrument.

As the selection of this, or any other artificial aid, must be a matter of individual preference the patient should be advised to try several before final selection, preference being given to that which conveys the voice most clearly with the least association of outside disturbing sounds. The subject of the modern electrical aids to hearing and of that important matter the reeducation of the hearing is one which would make a volume in itself[1] for the reeducation of hearing means something more than an increase of the special function, it is the reopening of a channel of stimulus to brain activity failing through desuetude.

The crowning disability of the chronically deaf is their tendency to isolation and to discouragement and this has to be met remedially by a distinct form of sociological work.

An experiment in this direction, the Speech Readers' Guild, so called because the furtherance of speech reading was the basis of its creation, has effectively made a centre for the otherwise isolated adult deaf and has accomplished much good not only educationally but by its inspiration. This association of a body of community members, much set apart individually, has already led to the formulation of a comprehensive study of the sociological status of the adult deaf with a view to the dissemination of information on this subject and the creation of other similar associations prepared to coöperate with all cognate activities but maintaining its creative independence.

The people whose needs have made the actuating impulse to this preliminary communication here presented, upon aid for the deaf, are, in the sense of the common weal, in virtue of the conservation of individual activity and in recognition of that humanitarian obligation which we are prepared to defend, the especial wards of such medical bodies as the American Laryngological, Rhinological and Otological Society, their number will be increased in consequence of the international struggle in which this country is now

(1) Dr. Thomas J. Harris. Reeducation of the hearing.

engaged and the appointment in this, and in similar societies in the United States of a permanent committee for the study of the needs of the adult deaf and the amelioration of their condition, with provision made for coöperation of the committees in question and collaboration with the Volta Bureau and the Reconstruction Service of the Industrial Union for the deaf, devoted to the welfare of the deaf, would be a national service.

# CHANGING METHODS AND ADVANCES IN THE TREATMENT OF PROGRESSIVE DEAFNESS FROM CHRONIC SECRETORY OTITIS MEDIA.

By FRANCIS P. EMERSON, M. D., Boston, Mass.

For the reason that otology is not an exact science, there are problems connected with its practice that should be reconsidered at stated intervals. One of these is the diagnosis, prognosis and treatment of that form of progressive deafness, which is the result of chronic secretory otitis media.

Previous papers on this subject were read before this Society by Dench in 1894, Harris in 1908, and by Holmes in 1912. These three papers are the only ones of record with the exception of Reik's paper on the relation of the tonsils to middle ear deafness.

The discovery of lymphoid hypertrophy in the vault of the pharynx, and the recognition of its relation to the eustachian tube by William Meyer marked an epoch in the prevention and treatment of this form of middle ear disease. This was fifty years ago. Following this discovery, the further appreciation of the close relationship of the nasopharynx to the middle ear made the organization of this Society a logical step. A large proportion of its present membership was educated first as otologists, and they have since studied and treated the nasopharynx only so far as in their judgment, such treatment, operative or non-operative could influence the prognosis of progressive middle ear disease.

In consulting the files of this Society, we find frequent reference made to the necessity of stopping infection by way of the eustachian tube. Hardly a writer, with the exceptions noted, has ventured to clearly define what he considered to be indicated along this line. The teaching in our Universities makes no attempt to correlate the two subjects of rhinology and otology in such a way that the student should know what to do, and what not to do rhinologically, to influence the middle ear. Each student is left to work out his own clinical experience, beyond instruction which relates specifically to the vault of the pharynx.

Observation among the leading otologists of today would lead us to infer that we are divided into two classes in our efforts to protect the middle ear. In the first class are those whose treat-

ment is almost wholly confined to the epipharynx and Rosenmüller's fossæ, to such an extent that it is common practice for their patients to consult them as aurists, and their confrere, as a rhinologist. In the second class are those who correct all deformities of the septum, abnormal conditions of the turbinates, drain the sinuses, remove tonsils, etc., and in fact do what any rhinologist would do, and believe, that in so doing they retard or check the progress of middle ear disease.

The writers of our latest text books give very scanty reference to remote foci, in discussing the etiology of chronic secretory otitis media.

In order that the writer might not misconstrue the attitude of the first group, namely, those who lay especial emphasis on the epipharynx, he sent the following question to fifteen prominent aurists. "Given a case of progressive deafness as the result of chronic secretory otitis media, where the hearing for the whispered voice has been reduced to 15/25ths, or less, would you expect any improvement from treatment of the nasopharynx, excluding the epipharynx and inflation?

Replies were received from ten, all of whom said they would not expect any benefit, provided the lymphoid tissue in the vault and fossæ had been thoroughly removed. One considered such a case as a beginning otosclerosis, and one had seen active operative measures do harm. In order to prove that positive improvement in hearing results from the cure of remote foci is not an easy problem, as the testimony of the patient, or of the aurist is of no scientific value, unless such observations are supported by careful hearing tests, and records covering a term of years, for we are dealing with a chronic disease, which is subject to acute exacerbations, and varying acuity of hearing.

Further, in order that such records should be available for analysis, such observations should extend over a period of from three to five years.

It would seem appropriate, after this length of time to consider again whether we have made any advance in the treatment of chronic deafness over the pioneer work of Meyer and Toynbee fifty years ago. Their work has since been elaborated by Politzer, Lucæ, Siebenman and others, but the essential facts are unchanged. Can we today offer any more hope to this unfortunate class of patients who comprise such a large part of our clientele? If some of them can be helped, is it possible to predict as a re-

sult of examination, that good results will follow our treatment in a given case, and how far any lost hearing may be restored?

In order that we may discuss this subject from common premises, let us define what we mean by chronic secretory otitis media. In a given case, otoscopic examination would show a dull grey, or ground glass appearance of the membrana tympani, absence of the light reflex, and retraction. The clinical picture would be one of slowly progressive loss of hearing, usually more advanced in one ear, with or without tinnitus. The hearing test would show a positive Rinné, the Weber lateralizing toward the more deaf ear. The low note by the Dench fork is unchanged, or slightly raised. The bone conduction is unchanged. The whispered voice, however, may be reduced to as low as 2 to 15/25ths. Given such a case, what conditions, if any, outside of the treatment of the epipharynx will influence its slowly progressive course? Whether any of the lost hearing can be restored is of the greatest importance to the patient, for the restoration of one or two notes in the upper register will determine to a marked degree, the efficiency of the individual.

In order that we may definitely promise ourselves, or the patient that the hearing will improve, the writer is guided by the following considerations, all of which depend on the question of reinfection.

It is his experience that in every case of progressive deafness from chronic secretory otitis media, there is a primary focus, which is constant for that individual. This may become quiescent, but in the interval it remains as a low grade process, subject to acute exacerbations. This point I wish to emphasize for until the primary focus is drained and cured, such acute exacerbations are a constant menace to the eustachian tube and middle ear. The course of the disease will be from bad to worse, and a little more rapidly if inflation is practised as a routine.

That there are pathogenic bacteria in the mouth and pharynx at all times is common knowledge. That they possess sufficient virulency under favorable conditions to cause serious pathological conditions in remote organs has been proven by the investigations of Miller, William Hunter, Billings and many other observers.

Depending upon the organism, and the virulency of the infection, the clinical picture is one of toxemia, septicemia or pyemia running an acute or chronic course. While this work is of recent date, all such facts had been observed years ago by Professor

Arkovy of Budapest from 1878 to 1898, substantiated by the painstaking microscopic findings of Miller of Berlin from 1884 to 1894, and by the clinical experience of William Hunter of London, who published his results in the Practitioner in 1900. Miller, who was educated as a physician, and also as a dentist showed bacteriologically that focal processes in the teeth, tonsils, or sinuses kept up a low grade infection of the adjacent tissues, and during exacerbations this might extend by continuity, or directly by way of the lymphatics, or blood stream to neighboring, or remote organs. That such low grade infection is constantly present either in the epipharynx, or quite as often at some other point in all cases of progressive chronic secretory otitis media, has been the observation of the writer for many years.

In order to determine where this primary focus may be, we inquire of the patient, in taking the history, where these acute exacerbations begin. It is a well known clinical fact that patients subject to acute infections almost invariably claim that for years such infections commenced in a certain manner, i. e., as a sore throat, and then went up or down or started as a head cold, and then went down.

In a given individual, this onset is almost constant, and does not usually alternate over many years, and indicates that the primary focus does not entirely subside, but becomes quiescent, and is subject to acute exacerbations, whenever the resistance of the host becomes lowered. This then gives us our first clue as to where we shall begin our treatment, and can not be determined by the anatomical conditions of the nares, even if a rhinitis be present. The next question is to ascertain if the hearing varies, i. e., whether it is better on some days than others. In such cases an active cause of the otitis is still present outside of the middle ear, and we can usually hope for improvement up to their best hearing, or more, if we remove the original focus, and clear up the accompanying infection.

Let us suppose a case where the history points to the acute exacerbations being more or less constant in the nose. It is the writer's opinion that no amount of anatomical deformity of the septum influences the course of chronic secretory otitis media until after infection takes place, but that such deformity predisposes to infection.

The tendency to a low grade infection in the nares is promoted by the following conditions. Given asymmetrical nares from devia-

tion of the septum and there results a compensatory hypertrophy of the turbinate on the concave side from over function from nature's attempt to equalize the air current. As a result, the soft tissue goes through the various phases of congestion, increased connective tissue and thickening of the mucosa. At this stage any acute infection, on account of defective drainage, may be continued as a low grade process subject to acute exacerbations. With the defective drainage, if a new infection is virulent enough, the sinuses may become involved. We therefore operate in the nares, not to remove a mechanical obstruction per se, *but to improve drainage and clear up the attending infection.*

To one who accepts this pathology, it is essential to the integrity of the tube and middle ear, that the infections of the nasopharynx should be cured so that acute exacerbations may be prevented. This can only be accomplished by improving the defective drainage, and equalizing the air pressure by reconstruction of the nasal chambers, i. e., straightening deflected septi, removing redundant tissue from the turbinates, reducing posterior hypertrophies, and opening all infected cells.

After this, it is equally important in cases of deafness of long standing, that no low grade process persists, as an epipharyngitis, or some focus be left that may still be subject to acute exacerbations, and thus offset the effect of our operative procedures as far as improvement of the eustachian tube is concerned. The middle ear condition grows progressively worse by reason of re-infection from the same primary cause, or from secondary foci, until the hearing is beyond improvement. For this reason auditory re-education by whatever means, inflation, electricity, etc., that does not take into account the underlying cause cannot be of permanent value until such focus is removed.

*Pharynx.* In the pharynx all lymphoid tissue, however small should be removed, including bands between the eustachian tube and the pharynx, and degenerate tissue in Rosenmüeller's fossæ. Where posterior rhinoscopy shows chronic congestion on the lateral pharyngeal wall, the condition of the fossæ cannot be determined without a digital examination. Chronic pharyngitis indicated by a glazed appearance of the posterior wall, and a change in the secretions, the writer has always found to be due to caries in the sphenoid, either alone, or in connection with a pyogenic focus in the posterior ethmoids; and the so called pharyngitis sicca he has not found to exist as an entity. While the diagnosis cannot be

made with an ordinary probe, it usually can be made by using one whose shank is no larger than a knitting needle, and that has a fused bead on the end.

*Tonsils.* The tonsils in their relation to local and systemic disease are only beginning to be appreciated, especially when found in adults between thirty and sixty years of age. As we now understand their pathology they account for many of the diseases that were supposed to be constitutional, such as scrofula, rheumatism, autointoxication, etc. Visual examination is useless. The anterior pillar should be pulled forward, and pressure used between the outer wall and the tonsil. In many cases where the tonsil is an active cause of the deafness, free pus will be demonstrated on one side, usually at the junction of the vela lobe, and the middle third of the tonsil, particularly in those cases showing toxemia. This is an enclosed abscess of streptococcus origin and subject to repeated acute exacerbations. The toxemia is marked, and it is probably the cause of chronic nerve degeneration in the labyrinth. Such cases are attended constantly by a low grade pharyngitis. Reik, in his article published in 1908 on degenerate tonsils, and middle ear deafness, has fully described their relation to the eustachian tube.

In the writer's experience the extension to the eustachian tube is more often by continuity and through the lymphatics and blood stream along the posterior pillar. The resulting low grade process can be seen not only on the lateral wall, but in the epipharynx, and over the surface of the posterior nares, as well as in the mouth of the tube.

*Eustachian tube.* The pathology of the tube in its relation to middle ear deafness, the writer has usually found either at the isthmus, or the pharyngeal end. Examination shows the tube, if the process has existed long enough to affect its lumen, as it usually has in *chronic cases,* more widely open on the side of the most affected ear, hence inflation is not only counter indicated, except for diagnostic purposes, but it makes the loss of hearing more rapid. Indeed, after stopping the infection, one of the next steps in our treatment is to try to restore the lost tone of the pharyngeal end by applications within the tube, and exercises that shall strengthen the function of the palatine muscles. The most experienced aurist will be deceived as to the lumen of the tube by auscultation, as very many cases have a decided narrowing at the isthmus when inflation seems clear. If no mechanical obstruction exists at either end of the tube, nature has provided for sufficient ventilation of the

middle ear. If such obstruction does exist in *chronic cases* inflation will do no good, and will add to the gradually diminishing function of the tubal muscle. Applications within the tube are of great benefit, but they should not be repeated too often, or be strong enough to excite too much reaction. This reaction, where the obstruction has been at the isthmus, has been one of the most difficult things for the writer to control in the treatment of the tube.

*The alveolar process.* The best work on the pathology of the alveolar process has been done by dentists. The relation of such foci to local and systemic disease has been repeatedly called to your attention by Dr. Haskin. Miller of Berlin in 1894 showed the seriousness to a patient with low resistance of pulpitis, grangrene of the pulp, pericementitis, alveolar abscess, ostitis, osteomyelitis, periostitis, alveolar necrosis and pyorrhea. He called attention to the fact that pyogenic organisms associated with osseous necrosis were very virulent, and that the soft tissues over a considerable area showed infection. His bacteriological findings proved that such local foci might not only involve adjacent tissue, but extend by continuity to the tonsils, and pharynx, or through the lymphatics or blood stream to distant organs. For our purposes such diseased conditions should be investigated in conjunction with a dentist, and all devitalized teeth examined by an X-ray, as part of our routine examination in locating foci that may be the cause of a progressive chronic secretory otitis media.

It is often asked why if such causes are active in the etiology of chronic secretory otitis media, it is not easy to settle the question by producing a series of cases corroborated by hearing tests. The answer is because of the fact that such foci are usually multiple and few men have systematically attempted to remove all sources of re-infection. Our knowledge of how to drain all the sinuses, of the menace of pyogenic foci in adult tonsils and the alveolar process, is too recent to enable us to tabulate a long series of *chronic cases* successfully treated. The improvement is necessarily slow and extends over considerable time. With energetic early treatment there is a more hopeful outlook for this field of otology.

As a line of investigation to be further developed, the writer sees no reason why such active foci as are constantly found in the teeth, tonsils and sinuses, should not contain bacteria with selective characteristics, i. e., in some we would have our pathology in the mucous membrane, in others the connective tissue, and in still others the osseous structures.

## CASE HISTORIES.

### Sinuses (Focus).

Dec. 2, 1911. J. F. W. Born, N. H. Age, 51 years. Single. Provision broker.

*P. H.* Measles at 21 years of age. Rheumatism three years. Tobacco moderately, no alcohol. Neiser infection. Repeated head colds. Teeth all removed. General health good, except for history of gall stones.

*Hearing.* Not normal for ten years. Worse for past 18 months. Sounds confusing. Sharp pain in both ears. Tinnitus. No history of aural discharge. Acute infections start in the head.

*Examination.* B. m. t. thickened. Light reflex gone, retracted. B. nares, pus in middle fossae. Adenoid tissue central in the vault. R. R. fossa obstructed. B. eustachian tubes open, the right being more open than the left.

*Diagnosis.* B. o. m. sec. ch. B. sup. ethmoiditis. Repeated reinfections from a pyogenic focus in the ethmoid sinuses.

*Treatment.* Exenteration of both ethmoid labyrinths. Argyrol, 20 per cent. to eustachian tube. Inferior turbinates trimmed. Nasopharynx cleansed with normal salt and sprayed with argyrol 20 per cent. until infection cleared up. *No inflation* except for diagnosis.

*Hearing tests.*

| *Dec. 2, 1911* | | | *March 15, 1913* | | | *March 13, 1915* | | |
|---|---|---|---|---|---|---|---|---|
| R | | L | R | | L | R | | L |
| 2/25 | W. | 16/25 | 15/25 | W. | 17/25 | 12/25 | W. | N. |
| 2/35 | acu. | 9/35 | 32 | D. | 32 | 30″/10″ | R. | 35″/13″ |
| 30″/10″ | R. | 35″/13″ | | (L. L.) | | | W. | |
| + | W. | | | | | 32 | D. | 32 |
| 32 | D. | 32 | | | | | (L. L.) | |
| | (L. L.) | | | | | | | |

| *May 13, 1916* | | | *Jan. 20, 1917* | | |
|---|---|---|---|---|---|
| R | | L | R | | L |
| N. | W. | N. | N. | W. | N. |
| 32 | D. | 32 | 35″/10″ | R. | 45″/10″ |
| | (L. L.) | | | W. | |
| | | | 32 | D. | 32 |
| | | | | (L. L.) | |

*Result.* No colds for a year. No tinnitus. Hearing normal.

### Tonsil (Focus).

May 20, 1915. Mrs. H. Born, R. I. Age, 59 years. Married. No children.

*P. H.* Scarlet fever and typhoid at 29 years of age. Pneumonia 18 months ago. La Grippe several times, but not severely until 1915. Tonsillitis annually for several years. Acute infections start in the throat. No aural history until five years ago, when the hearing became impaired beginning in the right ear. Occasional tinnitus. No climatic variations. Slight catarrh. Pyrosis. Ears worse after each throat irritation. Is below weight. Nose red, color poor, and has been a semi-invalid for twenty years. Never rugged. Prominent otologist told her that nothing could be done, and instructed her maid how to inflate the ears daily.

*Examination.* M. T. B. show ground glass appearance, dull, lusterless. L. nares. ant. deviation of septum, so that an applicator cannot be passed. Contact with inferior turbinate. M. M. not injected, and no infection present. Septum, sigmoid deflection.

*Sinuses* transillumninate normally, epipharynx negative. Pharynx, low grade pharyngitis. Tonsils, cryptic tonsillar disease. R. free pus, (one or two drops).

*Diagnosis.* B. o. m. sec. ch. Cryptic tonsillar disease. L. ant. deviation of septum. Progressive deafness secondary to tonsillar infection.

*Treatment.* Removal of tonsils, and after treatment until infection was gone. June 2nd, operation, Des Braisay Hospital. Complete tonsil enucleation.

*Hearing tests.*

| *May 20, 1915* | | | *Oct. 18, 1915* | | | *Dec. 10, 1915* | | |
|---|---|---|---|---|---|---|---|---|
| R | | L | R | | L | R | | L |
| 6/25 | W. | 6/25 | 4/25 | W. | 20/25 | 9/25 | W | 20/25 |
| 20″/10″ | R. | 32″/15″ | 20″/7″ | R. | 25″/11″ | 30″/15″ | R. | 22″/12″ |
| 512F. | | | 32 | D. | 32 | 32 | D. | 32 |
| W. | | | | | | | | |
| 32 | D. | 32 | | | | | | |

| *Jan. 11, 1916* | | |
|---|---|---|
| R | | L |
| 10/25 | W. | N. |
| 32 | D. | 32 |

*Result.* Pyrosis gone, weight increased. The right ear, which was the first involved, gained 4 feet. The left is normal. No inflation.

TONSILS (Focus).

June 4, 1916. Mrs. A. L. M. Born, Massachusetts. Age, 66 years. Widow.

*P. H.* No history, aural discharge or otalgia in early life. Hearing has been growing worse for ten years.

*F. H.* Patient has two children living. Husband died 21 years ago, cause unknown (suicide). One brother and one sister living and in good health. No family history of deafness.

*F.* Died age of 60, cause unknown.

*M.* Died age of 80, cause unknown.

*P. I.* Patient has had some ear trouble for the past ten years, which has slowly been getting worse. Hearing varies and is worse when she is tired, or has a cold. History of head colds and sore throats once or twice a year. Ringing tinnitus occasionally. Sensation of stopping of ears. History of rheumatism. L. hand shows swollen joint. Used to wake with a headache. L. tonsil shows cryptic disease. Pulsation in ears since sickness three years ago.

*Examination.* B. o. m. sec. ch.

*A. D.* M. T. indrawn and dull. *L. R.* gone, no marked thickening.

*A. S.* M. T. indrawn and lusterless. *L. R.* gone. Both tubes open but the right a little the more. Isthmus not narrowed.

*Treatment.* L. tonsillar infection cured by operation. No inflation at any time except for diagnosis. Local applications to tubes. Multiple and slow sinusoidal currents used for five minutes in each ear weekly. Current caused vertigo and nausea at first.

*Hearing tests.*

| *June 4, 1916* | | | *Dec. 15, 1916* | | | *Feb. 8, 1917* | | |
|---|---|---|---|---|---|---|---|---|
| R. | | L. | R. | | L. | R. | | L. |
| 1/25 | Whisp. | 2/25 | 3/25 | Whisp. | 3½/25 | 8 | Whisp. | 8 |
| | | | | | | — | | — |
| 10″/7″ | R. | 10″/5″ | | | | 25 | | 25 |
| | 256c | | | | | N. | U. L. | N. |
| ------- | Weber | ------- | | | | 32 | L. L. | 32 |
| 64 | L. L. | 64 | | | | | | |
| 1/10 | U. L. | N. | | | | | | |

*April 2, 1917*

| R. | | L. |
|---|---|---|
| 10/25 | W. V. | 9/25 |
| 13/7 | R. | 13/6 |
| ------- | Weber | ------- |
| N. | U. L. | N. |
| 32 | L. L. | 32 |

*Result.* Patient was gaining the most when she stopped treatment on account of the difficulty of coming into town at her age

## *Conclusions.*

1. Every case of chronic progressive middle ear deafness has a primary focus, which persists as a low grade infection, subject to acute exacerbations. In chronic cases such foci are usually multiple.

2. Such primary focus is usually constant for the individual, and is indicated by the location of exacerbations.

3. Every case showing variable hearing can usually be improved up to their best hearing or more.

4. So called cases of nerve deafness of non-specific origin are in the experience of the writer due to toxemia from some definite focus.

5. Inflation in *chronic cases* is unscientific and harmful as a routine, as the tube is already open and has partially lost it's tone in the majority of cases, and in those cases not open, it does nothing to remove the cause.

6. Nasal obstructions do no harm to the middle ear unless infection is present. Such obstructions, however, are the primary cause in the development of imperfect drainage, which predisposes to infection, and which is always present in cases of chronic secretory otitis media originating in the nose.

7. Foci whether in the sinuses, tonsils, mandible or epipharynx are potential factors in the progress of chronic progressive otitis media, either by direct extension or through the lymph and blood streams.

8. No hearing test will forecast the improvement in a given case as long as we have a positive Rinné with variable hearing.

9. Whatever the macroscopic appearance of the membrana tympani, the cause of the deafness is active for a long time outside the middle ear as a toxemia, or low grade infection subject to acute exacerbations.

10. Constitutional diseases have but little effect upon the course of chronic secretory otitis media, except to lower the resistance of the patient and make them more susceptible to exacerbations of their localized focus or foci.

# PRIMARY DISEASE OF THE LABYRINTH AS THE RESULT OF FOCAL INFECTION.

By GEORGE E. SHAMBAUGH, M. D., Chicago, Ill.

Accurate knowledge of the diseases of the internal ear has been very difficult to acquire, owing especially to the difficulty surrounding the study of the pathology of the labyrinth. In recent years a great deal has been done to clear up obscure facts relating to the pathology of those changes in the labyrinth which occur secondary to infection in the middle ear. The pathology also, in cases of long-standing nerve deafness, is now well understood. There still remains a great deal of obscurity regarding the pathological changes in the labyrinth, especially in cases of primary disease of the internal ear. Here we still find it necessary to piece out our knowledge by analogy from similar conditions occurring elsewhere. Fortunately our increased knowledge of the physiology of the labyrinth enables us now to interpret much more accurately than heretofore the symptoms arising from disease of the internal ear. Our improved methods for making a functional examination, moreover, enables us to detect more readily disturbances in the function of the labyrinth.

The clinical phenomena observed in these cases of primary involvement of the internal ear are quite definite, although the symptoms vary more or less widely in different cases. The defect in the hearing is always quite characteristic. It begins as a defect in the upper part of the tone scale, while the hearing at the lower part of the tone scale remains normal even after the defect for the higher notes has become quite extensive. In a few cases the loss of hearing does not begin at the highest part of the tone scale, but consists of a defect more or less circumscribed in the middle of the tone scale. For example, one may find a defect especially marked for the $C^4$ fork, while the Galton whistle may be heard normally throughout its entire extent. Paracusis Willisi is never a symptom in these cases, and tinnitus aurium, although often present, is rarely, if ever, the annoying symptom which it so frequently is in cases of otosclerosis. In most cases both ears become involved sooner or later and when both are involved the shortening of bone conduction in always present. In one-sided involvement the positive Rinné will

be changed to a negative, in those cases where the defect in hearing is quite marked.

Symptoms arising from an involvement of the vestibular part of the internal ear constitute a very important part of the clinical phenomena observed in many of these cases. Primary disease of the internal ear constitutes in fact the most frequent cause for the symptoms of vertigo. An involvement of the vestibular apparatus in these cases is, moreover, much more common than the occurrence of vertigo would indicate, as we have been able to ascertain since the introduction of tests for examining the function of the semicircular canals.

Another important clinical fact that has been brought to light by the introduction of the vestibular tests is that primary degeneration of the vestibular nerve occurs quite independent of a similar process involving the cochlear nerve. In these cases an occasional attack of vertigo is the only symptom indicating disease of the labyrinth.

We see, therefore, three distinct types of primary degeneration in the labyrinth: one where the cochlea alone is involved, producing nerve deafness and more or less tinnitus; the second, where the vestibule is involved simultaneously with the cochlea, producing, as a rule, occasional attacks of vertigo in addition to the symptoms arising from disease of the cochlea; the third type, where the vestibular nerve alone is affected, and where all symptoms indicating disease of the labyrinth may be absent, except for possible occasional attacks of vertigo.

The progress of this disease differs widely in different cases. Not infrequently there appears to be a gradually increasing degeneration of the parts involved. In many cases progress of the degeneration is accelerated by acute exacerbations. In others an acute attack is apparently followed by a long period of quiescence, when there is no progress in the degeneration, at least none that can be detected by our examinations.

The variations in the symptoms depending on the way in which the disease of the labyrinth progresses is especially striking for those cases where the vestibular part is involved. An insidious degeneration produces no symptoms excepting the gradual development of nerve deafness associated, perhaps, with some tinnitus. Where the degeneration is accelerated by acute exacerbations this is recognized by an increase in the deafness and the development of a more distinct tinnitus, and, where the vestibular part is involved, by the occurrence of distinct attacks of vertigo. The symptoms

arising from the vertigo sometimes quite overshadow the other phenomena. When the vertigo is severe enough to cause nausea and vomiting the cause of the disturbance is often mistaken for some gastric disorder. The actual disturbance produced by the vertigo varies from a slight disturbance of equilibrium to a profound shock, which throws the patient violently to the ground and produces dizziness which does not disappear for several days or even weeks. Where one has an opportunity of examining a case during the attacks of vertigo, one is able to recognize the characteristic vestibular nystagmus.

The question of the diagnosis of labyrinth disease is especially interesting in those cases where the principal symptoms are those resulting from the vertigo, such as nausea and vomiting. Before the introduction of the vestibular tests the diagnosis of a possible labyrinth origin for the vertigo was based on the presence of symptoms of disease of the cochlea, that is, tinnitus and nerve deafness. In those cases where a diagnosis could be made of a degenerative process going on in the cochlea, it seemed fair to assume a probable labyrinth origin for the vertigo, since it is known that primary degeneration in the labyrinth is the most frequent cause for this symptom. It is evident that in those cases where the degenerative process was limited to the vestibular nerve, that is, where the hearing was not affected, the labyrinth origin for the attacks of vertigo would often pass unrecognized. Fifteen years ago I began compiling a series of such cases where the symptoms complained of were those of vertigo and the disturbance caused by the vertigo, such as nausea and vomiting, but where there was no involvement of the cochlea and where no other cause for the vertigo could be detected. At that time it was necessary to assume a condition which we could not demonstrate, namely, a primary involvement of the vestibular apparatus, independent of any disease of the cochlea. In recent years, since the introduction of the vestibular tests, I have been able to go over a number of these cases and to demonstrate the loss of function in the semicircular canals.

The etiology of primary degeneration of the labyrinth is quite clear in many cases. It is not infrequently a complication of syphilis, both hereditary and acquired. It is observed also as a sequel of the infectious fevers, especially in mumps, typhoid, measles and scarlet fever. There remain, however, a large percentage of those cases of primary disease of the internal ear, where the etiology has not been accounted for. It is in these cases where I have sug-

gested focal infection as a possible cause. This conclusion was based upon the similarity between the manner in which the labyrinth involvement takes place and the involvement of other nerves where focal infection is known to be the cause. When the involvement of the labyrinth is associated with other evidences of systematic infection of focal origin, such as rheumatism or neuritis, one sees little difficulty in assuming a possible focal origin for the neuritis of the eighth nerve. In cases, moreover, where the degeneration of the eighth nerve develops quite independent of any other manifestation of systemic infection, a possible focal origin should not be overlooked, for it is not an infrequent clinical phenomenon to observe a single nerve the seat of persistent neuritis from focal infection.

I will report here briefly a few cases of primary labyrinth disease where focal infection appears to be the probable cause.

## CASE REPORTS.

The first case is that of a man, age 28, who consulted me in October, 1914. He had been subject to attacks of acute articular rheumatism since the age of ten, and had some joints which at the time of the examination were acutely involved. Evidence of an old endocarditis was also discovered. There was a history of recurring attacks of tonsillitis and although the tonsils had been operated they had been only partially removed. In February, 1917, the patient consulted me because of an attack of vertigo associated with tinnitus. The trouble had begun six days before as a tinnitus in the left ear, which he had noticed first on retiring. He was awakened in the middle of the night because of vertigo and nausea. The severity of the vertigo forced him to remain in bed for twenty four hours, and at the time when I saw him, six days after the onset, he was still annoyed by the disturbance of equilibrium. He recalled having had previous attacks of tinnitus but not of vertigo. He was still suffering from active rheumatism in several joints. A careful examination of the hearing failed to detect any defect. There was no spontaneous nystagmus, but the rotation test disclosed a marked depression of function for both the right and left labyrinth. The nystagmus following the rotation to the right, was extremely feeble and lasted but twelve seconds, and after rotation to the left was of the same character and lasted eleven seconds.

The second case is that of a man, aged 51, who consulted me in March, 1914, complaining of defective hearing, which he thought might have been coming on for several years. There was no tinnitus, no paracusis, and no history of deafness in his family. Examination disclosed normal drum membranes. A defect in the hearing for the whispered voice was found in both ears, especially marked for numbers like 66, which was heard only at one foot for either ear. The C fork, 64 d. v., was heard normally for both ears, as was also the C4. The Galton whistle (Edelmann) was heard at four in the right ear and at nineteen in the left.

The patient returned to the city again Dec. 10, 1914, when he complained of a high-pitched continuous tinnitus which had developed during an acute head cold one month before. There was a distinct increase in the defect for the

Galton whistle, especially in the left ear. The faucial tonsils were somewhat enlarged and distinctly congested. Cheesy plugs were expressed from both tonsils. There was no accessory sinus trouble and no infection about the teeth. In April, 1915, the patient discovered that he was running a temperature ranging as high as 100° and 101° each day. At one time it registered 103°. Examination of the blood showed a leucocytosis of 14,000. During this time he developed a severe persistent arthritis in the instep of the left foot, which came on spontaneously and was not the result of traumatism. There were no other rheumatic symptoms. This condition continued without abatement until June, when the tonsils were removed, after which there was a gradual subsidence of the temperature and disappearance of the leucocytosis.

The patient was seen again Dec. 6, 1916, complaining of increasing annoyance from the tinnitus in the left ear and from beginning tinnitus in the right. His general condition at this time was good and the arthritis had practically disappeared. The functional examination of the ears was carefully gone over. There was a distinct shortening of bone conduction and the Rinné was positive in both ears. The Galton whistle was heard at six in the right and 25 in the left ear. There had been at no time any symptom of vertigo, yet the nystagmus after rotation to the right lasted but nine seconds and after rotation to the left but six seconds and was very weak. The past pointing after rotation to both the right and left was still present but very slight.

In this case the chief symptom was the insidious development of nerve deafness, which became quite extensive in the left ear. Tinnitus developed after an acute infection only after the condition had been present several years. At no time was there any vertigo, yet the examination disclosed a very marked degeneration of the vestibular nerve in each ear. The patient's general condition remained good except for a period of several months in 1915, when he was running a temperature. There was no history of syphilis and the Wassermann test was entirely negative. The patient has suffered from no infectious fever but he did suffer from distinct evidence of a systemic infection, as shown by the protracted rise in temperature, the leucocytosis and the arthritis. It seems logical to assume that the same cause was responsible for the degenerative neuritis which involved both branches of the eighth nerve.

A third case is one where the neuritis of the 8th nerve followed distinctly from an attack of acute tonsillitis. The case is briefly as follows: Mrs. H., aged 34, consulted me May 15, 1917, especially because of vertigo and deafness. She gave the following history of the onset of her trouble: Two weeks ago she contracted a severe tonsillitis on the left side, which developed with white patches. No cultures appear to have been made. She suffered from severe headache for three or four days. During this time she developed facial paralysis on the left side and huskiness of the voice, which examination showed was due to paralysis of the left vocal cord. At the same time she began to suffer from slight tinnitus in the left ear, which resembled the noise of a sea shell and was associated with a defect in the hearing and intense vertigo, with nausea and vomiting.

When she consulted me I found the facial paralysis was still practically complete on the left side and the left vocal cord was immovable. Drum membranes were quite normal. Transillumination of the mastoid showed normal condition both sides. Functional examination showed the hearing in the right ear to be entirely normal. In the left ear the whispered voice was heard only

close to the ear and it might easily have been perceived by the opposite ear. The hearing for the C fork, 64 d. v., was only slightly shortened in the left ear. The Weber was lateralized to the right. The Rinné test was positive in both ears. The hearing for the C4 fork was only slightly shortened in the left ear, while the hearing for the Galton whistle was but slightly shortened at the upper limit. A striking peculiarity in the hearing of the left ear for all the tuning forks was that they were heard distinctly as at least an octave higher in the left ear than in the right and this applied to the Galton whistle as well. The Romberg was positive, the patient falling to the left. There was a spontaneous rotatory nystagmus, which still persisted especially strong on turning the eyes toward the left, but disappeared entirely on looking to the right. Spontaneous pointing showed slight deviation to the left. Nystagmus after rotation to the right was very weak and lasted but seven seconds; to the left was quite strong but disappeared in about twenty seconds. Caloric stimulation produced no response in the left ear but normal response in the right ear.

*Diagnosis.* Peripheral toxic neuritis, as the result of tonsil infection involving the eighth nerve, the seventh nerve, and responsible for paralysis of the left vocal cord. The case was examined carefully by Dr. Thor Rothstein, a neurologist, who arrived at the same conclusion.

The last case to which I will refer was a Mrs. K., aged 61, who consulted me in May, 1917, because of roaring, tinnitus aurium, deafness and attacks of vertigo, which developed suddenly January last. She has had a number of attacks in which the vertigo is associated with nausea and vomiting. The patient has been subject to recurring tonsillitis for many years. The tonsils were found to be enlarged, congested, crypts filled with cheesy deposits, in other words the seat of a chronic infection.

In the functional examination the whispered voice was heard normally in the right ear; in the left ear there was a distinct shortening for 66, but not for 18. Weber was not lateralized. Schwabach test showed distinct shortening of bone conduction. The Rinné was positive in both ears. The C fork, 64 d. v., was heard normally in the right ear and only slightly shortened in the left. The same was true for the C4 fork. The Galton whistle showed a distinct defect at the upper end of the tone scale, involving both ears. Examination of the vestibular apparatus showed spontaneous pointing normal. There was no spontaneous nystagmus when the patient was examined. Rotating the patient to the right developed a nystagmus which was very strong with rapid movements and which lasted 32 seconds. Rotation to the left developed a nystagmus which was very weak and lasted only 16 seconds. In this case there is apparently a neuritis involving both sides but with the cochlear nerve more involved on the left side and the vestibular nerve more involved on the right.

## DISCUSSION.

**Dr. Max A. Goldstein,** St. Louis, Mo., thought the consensus of opinion very well reflected in the two papers, leaving very little room for difference of opinion save concerning minor points. He took issue with Dr. Emerson as to the nomenclature and the term chronic progressive middle ear deafness. The functional tests now available made it possible to differentiate between chronic catarrhal and chronic otosclerotic processes. Focal processes in the middle ear could be reached from the nasopharyngeal end of the tube. The treatment of otoscler-

osis in any stage showed little if any definite effect so far as the end results were concerned. He doubted if sufficient progress had been made with reference to the spongifying process or fixed end of the stapes to say anything about therapy. However discouraging it might be, the fact remained that very little value could be attached to any form of treatment in clearly defined, clinically differentiated otosclerosis. He could not refrain, in the light of this doubtful state of affairs, from emphasizing the importance of giving attention to the education of the patient in efficient lip reading.

**Dr. Hanau W. Loeb,** St. Louis Mo., concurred in what Dr. Shambaugh had said concerning the likelihood of a focal origin of these obscure symptoms, which might be called vertiginous symptoms, so often referred to the labyrinth. Abscess at the roots of the teeth, in his opinion, was more apt to be the cause than infection of the tonsils. He had had several cases of this type. In one the seventh nerve was affected, with spontaneous nystagmus and deafness. Facial paralysis, nystagmus and then deafness, was the order of disappearance of symptoms. The teeth were the cause in this case. The subject presented by Dr. Shambaugh was a very important one, and would doubtless, in time, help to clear up the cause of Ménière's disease, disturbances of the eighth nerve, and other obscure conditions.

**Dr. Henry Horn,** San Francisco, Cal., thought it quite significant that the two essays were grouped together for discussion. He ventured to prognosticate that within five years the condition considered by Dr. Emerson, otosclerosis, and all other middle ear affections, would no longer persist as clinical entities. The trend of opinion was leading more and more clearly to the conclusion that middle ear disease is always accompanied by more or less involvement of the labyrinth. The examination of recruits for the aviation service would give otologists an opportunity to examine an enormous number of normal individuals in an absolutely uniform and methodical manner. The conditions under discussion had been obscure because of the lack of a methodical manner of making the tests. The diagnosis, therefore, had been largely a matter of the examiner's personal equation. It had been brought out recently, in Philadelphia, that a large number of otherwise absolutely normal individuals had some involvement of labyrinth. The conclusion would finally be reached that focal infection is the cause of everyone of these diseases.

**Dr. George W. Mackenzie,** Philadelphia, Pa., directed attention to the fact that there might be quite a number of conditions referable to the ear that might cause tinnitis, vertigo, nystagmus and equilibrium disturbance—(a) congestion of the internal ear manifested by increased nystagmus to the side of the lesion, together with vertigo and past-pointing; (b) there might be also a plastic condition secondary to chronic middle ear suppuration; (c) a spongifying process might, in its turn, invade the osseous labyrinth; (d) labyrinthitis of the circumscribed or diffuse type; (e) nerve lesions following infectious fevers,—the post-infectious type of neuritis; (f) a granulomatous inflammation; (g) toxic inflammation due to lead, alcohol, etc.; (h) retro-labyrinthine inflammation leading to primary atrophy, in syphilis; (i) neuritis, of the so-called refrigeratory type from a draft of cold, damp air. Cases of this type had been studied microscopically, and a round-celled infiltration found, but without microorganisms. Doubtless many of the cases reported really came within this class. By what test could one differentiate between a lesion in the nerve and one in the internal ear during life? The galvanic test was the one only test for the differentiation between inner ear and nerve lesions.

**Dr. Emerson,** in closing the discussion, said he had tried to define the type of middle ear under discussion, stating that in this type there

was no change in bone conduction, in the promontory, or in the low notes, and that they were therefore in the category of cases of chronic secretory otitis media.

**Dr. Shambaugh,** in closing the discussion, said that otologists were fairly well satisfied as regards the treatment of the suppurative cases of chronic otitis media, but these formed only a small part of the chronic middle ear cases. The treatment of the non-suppurative cases through the Eustachian tube accomplished much more for the acute and sub-acute conditions than it did for the chronic cases. Long continued treatment of a chronic case via the tube, that is by inflation, not infrequently resulted in a permanent injury due to over-stretching of the drum membrane. It was much more important that a careful search be made for the foci of infection in the upper respiratory tract which acted as the underlying cause for the changes in the middle ear.

In cases of otosclerosis heredity seemed to play a very important part, and yet an inherited tendency to this condition might remain latent in an individual until it was stimulated and brought out by some infection. An analogous condition was seen in cases of chronic arthritis deformans. There appeared to be not infrequently a hereditary tendency and yet the process was undoubtedly stimulated into activity by some focus of infection.

# SURGICAL PATHOLOGY OF THE MASTOID.

By JOSEPH C. BECK, M. D., Chicago, Ill.

The surgical pathology or more correctly the pathological anatomy of the mastoid process takes into consideration only a definite anatomical area although this is probably never solely involved. Usually the middle ear and at times the internal ear are associated in the pathological process. Neither of these later conditions will be considered in this paper.

The pathologic changes are subdivided in the first place into gross or macroscopic and minute, histologic or microscopic. A further division is absolutely essential and that is acute, chronic and acute exacerbation of a chronic disease. The anatomy of the mastoid process, the bacteriological elements, the general condition of the patient as well as his management during an attack of mastoid disease will determine the pathological changes. The discussion of these factors are not within the title of this paper. but I would like to say that the roentgenogram, the laboratory, the medical team-mate, and hospital practice will go a long way to modify these pathological changes.

## Acute Mastoiditis.

*Gross Changes.* I have divided these into two great classes, based upon the study of a sufficient number of cases operated upon and the examination of ninety-six microscopic sections. These I have designated (a) Cell route or confluent mastoiditis, (b) Vascular route or osteophlebitic mastoiditis of Grunert.

In the former we usually find an external fistula over the antrum or near the tip, with considerable infiltration of the periosteum. The bone bleeds easily and at times appears darker due to edematous and engorged mucous membrane under the thin cortex. As soon as the latter is removed, a fair quantity of pus escapes, depending upon the presence of a fistula and the type of microorganism present. The bleeding is usually very free (to a novice it may appear as though the sinus were opened) and the lining membrane usually protrudes through the opening made. The intercellular septa are usually broken down either over the antral region or the tip, and at times both. Not infrequently in this confluent form there is exposed an area of the lateral sinus,

digastric fossa or dura. The histologic changes in these structures will be shown later by means of micro-photography. In the vascular route or osteophlebitic type of acute mastoiditis, we find macroscopically the tissues overlying the cortex very little changed. The latter bleeds very little if any, and has otherwise a fairly normal appearance. On opening the mastoid we find the bone, however, very red. There is little or no pus escaping. The cells are well preserved, and their lining membrane is not very edematous, in contradistinction to the cell route type of infection. The preserved intercellular septa on close inspection shows the marked engorgement spoken of above. Only in a later stage, in which a possible secondary infection takes place, can one find any exposures of the lateral sinus, although the same may have an infection about it or within it. It is this form of mastoiditis that has the greatest percentages of perisinus abscess and sinus thrombosis developing, due to the extension by the venous infection from the bone. The same is true as regard the erosion of the other vital neighboring areas, such as the dura of the cerebrum or cerebellum, the labyrinth, the facial canal and the digastric fossa. The histologic changes of this type of affection will be described later, by the aid of microphotographs.

### Chronic Mastoid Disease.

*Gross Changes.* From the study of a sufficiently large amount of material of operated cases, and the subsequent microscopic examination of particles of mastoid bone removed, I have been able to identify or classify the changes as the following:

1. Osteofibrosis or sclerosis.
2. Osteofibrosis with fistular tracts.
3. Osteofibrosis, fistular tracts and cholesteatomatous infiltration.
4. Osteofibrosis, fistular tracts, cholesteatomatous infiltration with cavity formation containing cholesteatomatous masses.
5. Tuberculous osteitis.
6. Syphilitic osteitis.
7. Actinomycotic osteitis.
8. Repairative osteitis.
9. Foreign body in mastoid, (a) sequestrum, (b) any other substances.
10. Neoplasms. (a) Sarcoma, (b) Carcinoma, (c) Endothelioma.

GROSS PATHOLOGY.

(1) *Osteofibrosis.* As the name implies the mastoid bone is converted into a more or less solid bone. The cortex is hard and bleeds very little. The cells are conspicuous by their absence and oftentimes not a single cell is found until the antrum is reached. If any cells are present, they are very small and are located in or near the tip. The degree of sclerosis depends a great deal upon the form of mastoid, whether pneumatic or diploic. That is to say, that in the latter form osteofibrosis is easier and more complete than in a large celled mastoid. The histologic findings of most of the chronic conditions described in this paper will be shown later by means of microphotographs.

(2) *Osteofibrosis* with fistular tracts. Practically the same findings are encountered as in the preceding form except that a few cells are met with, and a number of tracts lined with granulation tissue. The bleeding is also more pronounced. In these cases there is frequently found an exposure over the promontory of the horizontal canal as well as the tegmen, a condition well to remember in the acute exacerbations, as causes of brain abscess, meningitis and labyrinthitis. The facial canal, and bony lateral sinus wall and other portions of the labyrinth are less frequently the seat of these necrotic areas. These localized necrotic areas are usually covered with granulations and it is considered bad practice to remove them as they serve as protection to further septic invasion.

(3) *Osteofibrosis, fistular tracts and cholesteatoma.* Again the same pathological changes are found as in the two varieties previously mentioned and in many cases the cholesteatomatous changes are not possible of detection grossly in the mastoid, but are microscopically. However when the antrum, aditus and attic are exposed by operation one will be able to remove a considerable mass of cholesteatomatous material. All are familiar with the characteristics of these masses, but they can be further identified grossly by the Bruehl test, by adding a few drops of chloroform to a mass when it will turn a yellowish green (cholestrin). The microscope will always demonstrate the characteristic crippled epithelial cells.

(4) *Osteofibrosis fistular tracts, cholesteatoma with cavity formation.* The cavity that is found in these cases varies in size from that of a small marble to the size of the mastoid process and extends in some cases beyond its confines. The location of

this cavity may be at the tip, over the antrum or continuous with the attic to the antrum, the socalled spontaneous radical mastoid operation. Pressure atrophy of the bone due to the continuous formation of epithelium in layers like an onion, plus the necrosis due to the infection is the explanation of this cavity formation. These masses often contain particles of necrotic bone (bone dust). The cavity itself is lined by a glistening membrane known as the matrix from which the new epithelial cells form. This matrix is not a mucous membrane or skin, not even an epithelial cicatrix.

(5) *Tuberculous osteitis of the mastoid.* Grossly, this condition cannot be identified, except that it may be suspected when the fistular tract formation is very marked, even finding one or more on the cortex, which is uncommon in other forms of chronic otorrhea. Again there are often softened areas of considerable size surrounded by very dense bone. Granulations are also more numerous throughout the mastoid process. Actual collections of pus are present in which, however, I have never found the bacillus tuberculosis.

(6) *Syphilitic osteitis of the mastoid.* This is as a rule a sclerosed process but not eburnated. At times it is entirely softened containing sequestra of considerable size. The granulations surrounding such a sequestrum are large and flabby and do not bleed very much. The three cases of this type which I have identified with subsequent microscopic examination all had the characteristic of having very little calcareous material remaining in the bone.

(7) *Actinomycosis of the mastoid.* Two cases of this on record, one by Majochi and the other I had the pleasure of studying under my service with Prof. Zanfal and the pathologist Prof. Chiari. We found the mastoid bone including the cortex practically riddled with fistulae containing thick pus in which there were the characteristic yellowish bodies (actinomyces). The bone was quite soft otherwise.

(8) *Reparative osteitis of the mastoid.* The pathologic process refers to the reoperated cases. The previously exenterated cavity is either filled with granulations, cholesteatomata or both, or it is lined by the characteristic epithelial scar. The bone of these cavities is as a rule very hard and the edges or margins of said cavity are thickened and irregular. It always appears (especially if one does the reoperating on his own case) as though the bony

cavity was much smaller than when it was made at the previous operation. Bleeding is very free from these granulations as well as from the bone. There can scarcely be a description of the various parts of the mastoid since each case is an entity of its own.

(9) *Foreign body in mastoid.* (a) The *sequestrated* type is, except in the syphilitic osteitis, already described, most frequently met with in children, usually the result of an incomplete simple mastoid operation, especially in the cell route or confluent variety of mastoiditis. These sequestra are always surrounded by somewhat healthy granulations, bleeding very freely. The sequestrum is usually very easily dislodged, is irregular in shape and has the appearance of being the axial part of several mastoid cells. At times these sequestra are very small, flat, with very pointed extremities. (b) As to other foreign bodies, they may be, shots, bullets, shell fragments, parts of instruments, such as gouges, knife and scissor blades. Surrounding such foreign body is usually an infected area with or without granulations, depending upon the length of time that the foreign body has been in the mastoid.

(10) *Neoplasms of mastoid.* I have had all the three varieties, sarcoma, carcinoma and endothelioma, but they were always the results of these growths in the vicinity of the mastoid. In the case of sarcoma, it was a retromaxillary tumor that finally involved the mastoid process. In the carcinoma, it was the result of a progressive epithelioma of the external ear, and in the case of endothelioma it followed a primary growth of that nature from the middle ear. The gross pathology was that of a malignant disease of bone anywhere in the body, except that it was complicated by secondary infection. The microscopic changes are also those characteristic of sarcoma, carcinoma and endothelioma. The type of endothelioma in this particular case was endovascular.

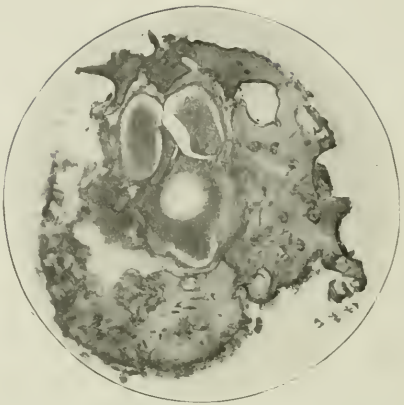

Fig. 1. Cell route infection. Three cells have coalesced and filled up with pus and edematous lining membrane. The greater portion of the bone is necrosed and a small portion is fairly well preserved bone, although much inflammed.

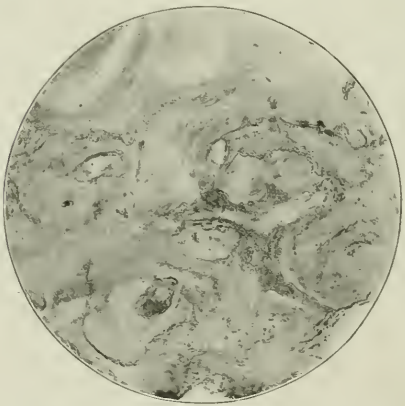

Fig. 2. Cell route infection. Practically every cell is filled up with edematous material and pus. The bone is involved in a cloudy swelling but not necrosed.

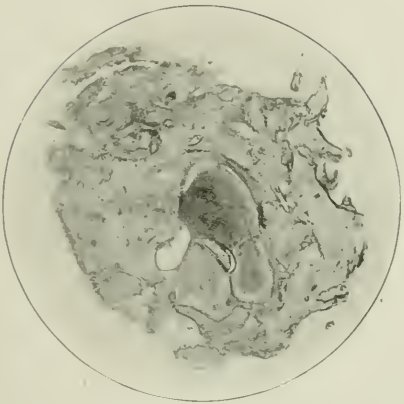

Fig. 3. Cell route infection. Practically the same changes as in Fig. 2 only more advanced in the coalescence, with cells filled with pyogenic material.

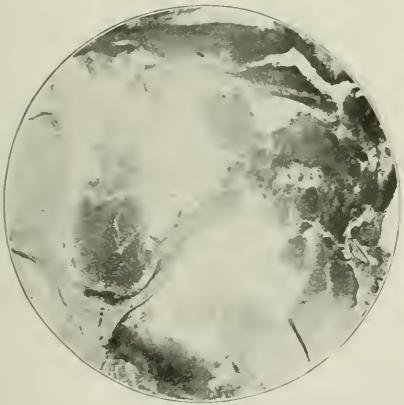

Fig. 4. Cell route infection. Markedly advanced case of necrosis and abscess formation, only a sliver of bone left here and there

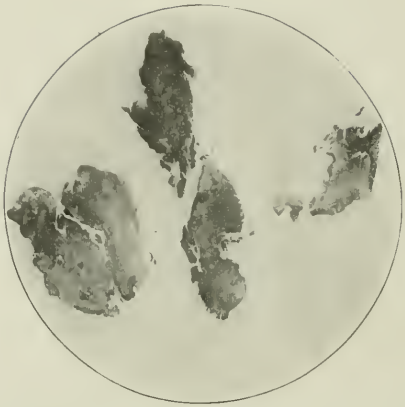

Fig. 5. Curetments of the interior of mastoid in cell route infection. The bone is necrobiotic probably beyond recovery.

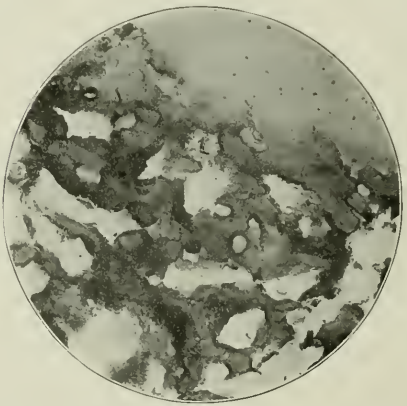

Fig. 6. Vascular route or osteophlebitic infection. The cells are well preserved and empty. The bone is highly inflammed with minute necrotic spots all through the section.

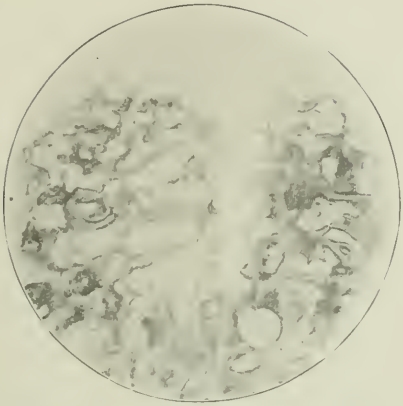

Fig. 7. Vascular route or osteophlebitic infection. The same process as in Fig. 6 only the bone is more necrosed.

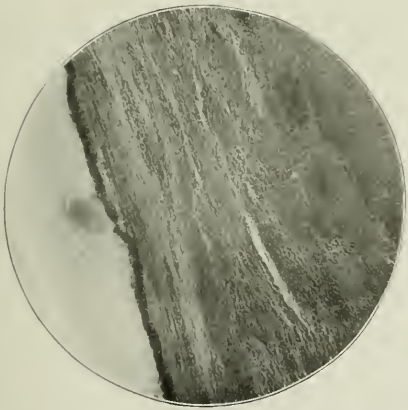

Fig. 8. Osteofibrosis. The bone is changed into fibrous tissue. There is no evidence of any recognizable bony structure, or necrosis.

Fig. 9. Osteofibrosis with fistulae formation. There are evidences of necrosis with tracks lined with granulations and pyogenic material.

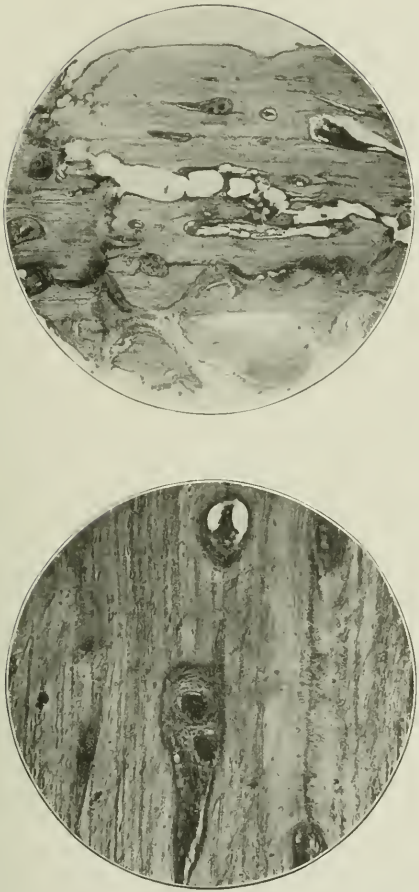

Fig. 10 a and b. Osteofibrosis, fistulous tracts and cholesteatomatous infiltration. The epithelial masses have a center of pus and show the spreading of the scarred bone.

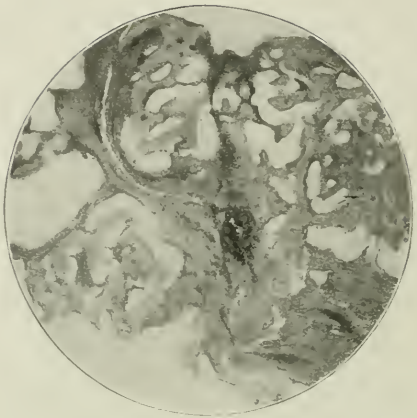

Fig. 11. Tuberculous osteitis with fistulae. The infected area is surrounded with subacute swollen bone. The center shows evidences of caseation.

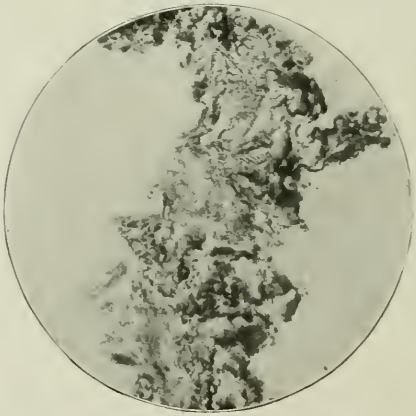

Fig. 12. Syphilitic osteitis. The bone is soft in areas and fibrous in other places.

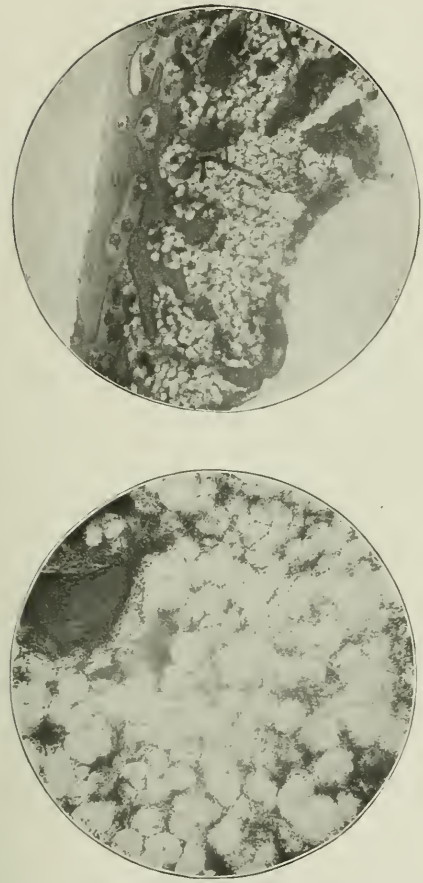

Fig. 13 a and b. Repairative osteitis. At the margin is seen fairly healthy spicules of bone from which is seen young osteogenic structures loosely arranged with a large number of small capillary vessels.

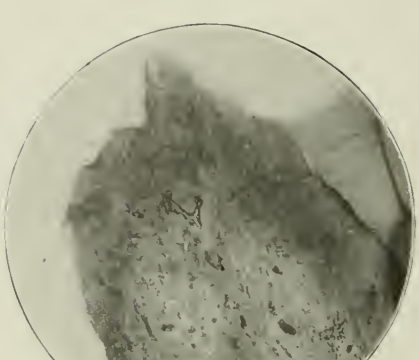

Fig. 14. Sequestral ostcitis. The characteristics of dead bone with a sort of a worm eaten appearance. About the margin is seen a frail granulation tissue and the jagged edges are very characteristic.

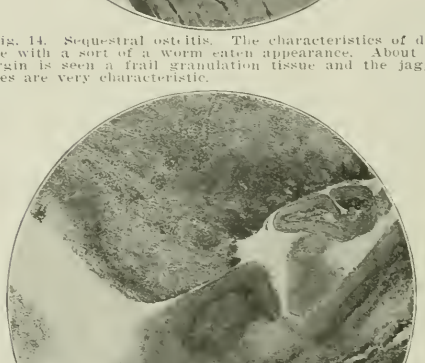

Fig. 15. Simple granuloma of antrum with folded up epithelial mass.

The arrangement of the epithelium suggests another possibility of cholesteatomatous formation. The granulation tissue is as any other of that type.

Fig. 16. Epidermal scar of radically healed mastoid cavity.

The conspicuous absence of any blood vessel or any resemblance to true skin. A much attenuated epithelial layer upon a firm scar is all that can be seen. The uppermost top layer of epidermic is necroblotic.

## DISCUSSION.

**Dr. George E. Shambaugh**, Chicago, Ill., emphasized the point brought out in this paper of Dr. Beck's that in suppurative conditions which involved the mastoid the changes in the bone were not always the same. The clinical problem which confronted one in the various cases of suppurative otitis media was quite varied. In the acute cases not infrequently there was no external evidence pointing toward a dangerous mastoid condition, and the indication for interference was based largely upon the presence of such symptoms as pain. In other cases one saw very marked external evidences of a mastoiditis, such as alterations of the outer surface of the mastoid, where one was quite justified in deferring operative measures and where the condition got well spontaneously. He had gathered the impression that cases of acute mastoiditis were handled quite differently in different parts of the country. In some places there seemed to be a more or less general tendency toward early interference where there was the slightest evidence of a mastoiditis. If there was one fact that was established in otology it was this: that external evidence of a mastoiditis is not in itself a positive indication for operative interference. Take, for example, the mastoid reaction which one observed so frequently, especially in children in the early stages of an otitis media, before the drum membrane was ruptured. Here one could have the auricle pushed away from the side of the head from edema and yet in the vast majority of these cases an incision of the drum membrane was all that was required.

In chronic suppurative otitis media one was confronted again with the question, which was not always easily determined, whether the situation was one which required surgical measures or whether it was one which could be safely treated locally. The experienced otologist had today little difficulty in differentiating between these two types, and the fact that this differentiation could be made obviated the necessity of operating unnecessarily on a great many benign cases of chronic otitis media.

**Dr. George W. Mackenzie**, Philadelphia, Pa., had been particularly interested in the fibrosis or sclerosis cases. It was surprising to find how small the antrum and how hard the mastoid was in cases of chronic middle ear suppuration. When healing failed to take place in acute middle ear suppuration, one thought of some obstructive or inflammatory condition in the nose, a complication in the tube, or in the oropharynx. In another class of cases the condition tended to become chronic after the correction of these faults. In those cases it was well to look for trouble in the mastoid. The worst cases of mastoiditis were those in which, anatomically, the external cortical layer was very thick. When the suppurative condition was found behind the aditus ad antrum it could not be reached by the anterior route; it was then necessary to employ the posterior operation by way of mastoid operation.

**Dr. Beck**, in closing the discussion, said most of the cases were of the osteo-fibrotic type. These cases, examined post-operatively, showed no trouble in healing. Osteo-fibrosis was present in practically all of them, but the process should be classified as to whether it was tuberculosis, syphilis, or what not.

# THE NEW USEFULNESS OF OTOLOGY.

By EUGENE RICHARDS LEWIS, M. D., Dubuque, Iowa.

Otology today finds itself in position to render services analogous to those which have brought merited recognition to ophthalmology. Aside from relieving ills strictly confined to the ocular tissues themselves,—refractive errors, muscle imbalance, cataract and the like—ophthalmology is requisitioned by men in other fields of medicine for valuable information concerning intracranial conditions, renal and vascular disease, metabolic disturbances and various intoxications. As a result of this sort of service ophthalmology has for years taken its proper place among the men in general medicine.

How about otology?

Does the internist turn to otology for accurate diagnosis of vertigo? Does the neurologist catalog among his stock dependabilities the accurate data otology can furnish concerning the VIII nerve and its intracranial connections? Does the general surgeon before operation eliminate by exclusion the toxic labyrinthitis whose symptoms may simulate obscure gall-bladder disease? Does the cranial surgeon avail himself of the additional aid offered by testing out certain pathways by ear stimulation in determining the operability of intracranial new growths? Does the syphilologist use the quantitative determination of responses to VIII nerve stimulation in diagnosing early luetic involvement of the central nervous system?

They do not.

They are all agreed as far as the ear is concerned that it is an organ to be heard *with:* they must be brought to realize that the time has come when the ear is also to be heard *from.* Today we see the internist and the surgeon awaiting the report of the ophthalmologist. Tomorrow they will await the report of the otologist as well.

The integrity of the vestibular apparatus is susceptible of such precise determination that the information derived from ear examination even surpasses that obtainable from the eye. There is much of otology not yet understood, but there is so much that we do understand *sufficiently to put it to good use* that it behooves us

all as otologists to apply ourselves to the work of disseminating this new usefulness to the utmost.

Jones and his associates have shown definitely that there are separate and distinct paths from the horizontal and from the vertical end-organs to the *same brain centers*. Impulses from the horizontal end-organ traverse the VIII nerve to the medulla where the pathway divides, one branch going to the center for sensing motion in the cerebrum, the other branch going to the eye-muscles. Their arrival is signalized in the cerebral center for perception of motion by consciousness of motion; in the eye-muscles, by a pulling of the eyes to one side. Therefore, if on applying a stimulus, the proper perception of motion and pulling of the eyes result, we can say definitely that that line is in working order, just as can the tester on a telephone line. The same is true of the vertical canals.

In applying the ordinary tests, caloric or turning, to a normal case, the first result of stimulation is a false sense of turning which is called "vertigo." Seated quietly in a turning-chair with closed eyes there is no disturbance of endolymph quiescence and the information reaching the brain from the end-organs notifies the individual that he is still. Now on revolving the chair to the right, the canals turning and the fluid within lagging by reason of inertia, interaction between cupola and hairs of the end-organs occurs, discharging impulses to the brain which bring to it the information that the individual is turning to the right. As the turning continues the fluid also gradually takes up the motion, and before long the canals and the fluid are in motion at the same rate of speed. At this state there is no discrepancy between the motions of the canals and the fluid, nor was there any discrepancy between these two when both were still prior to the turning. The information reaching the brain at this stage is the same, therefore, as when both were still prior to the turning, namely, that the subject of the turning is *still* again, *although this is not the fact*. Now if the turning be suddenly stopped the fluid continues flowing in the same direction, again stimulating the end-organs, but in the opposite manner, and the brain receives the information that the body is again turning, but this time in the opposite direction, namely, to the left. This also is not in accord with fact. The information received by the brain at the beginning of turning was not *vertigo* because it was *normal sensing of motion*, exactly in accord with fact. The subse-

quent information *was* vertigo because it was sensation of motion *not* in accord with fact. Therein lies the only difference between normal sensing of motion and vertigo.

There are several ways in which vertigo can be tested and comparatively measured. The one usually employed is the pointing test. With eyes closed, the normal individual can touch a fixed object with any convenient portion of his extremities, such as the index finger, the great toe, the forehead, withdraw as far from the object touched as possible and go back and touch this object again, *without* the guidance of vision. For convenience let us consider only finger pointing. The examiner's index finger is touched by the subject, or let us say by you, with closed eyes. You then raise your arm high, and go back to find the examiner's finger again. You will experience no difficulty in finding it, guided by your memory of where it was before the arm was raised. If this is attempted after turning, *during the stage of vertigo*, your finger will not find the examiner's finger but will reach out where you *think* it is. As we have seen, the brain is receiving the information that you are turning *to the left*, so you will *think* you have turned way past where the examiner's finger *was* when you lifted your finger off of it, and you will "past-point" to the right. The reverse of this of course is your experience when you have been turned in the opposite direction.

Another test of vertigo is the fall tendency. Feeling you are falling to the right (when you *are not*) naturally causes involuntary pull to the opposite side to counteract the falling you feel. The result is a fall to the left; similarly, feeling you are going forward, you fall backward, as the result of involuntary compensation. Thus does the fall tendency furnish accurate information concerning vertigo.

Now to consider the other effect of vestibular stimulation, nystagmus. The impulses from the horizontals cause the eyes to be pulled to one side in the horizontal plane, those from the verticals pulling the eyes to one side in the vertical plane. During full anesthesia this pull is manifest, whereas obviously the other motor phenomena connected with these tests are not—namely, past-pointing and falling. This eye movement is therefore *the one and only movement* of the body caused by impulses from the vestibular end-organs. Except during unconsciousness, the *pull* of the eyes is followed by a quick *jerk* in the opposite direction, the combination of the *pull* and *jerk* being so-called vestibular nystagmus.

Vertigo and nystagmus are the only *direct* responses to vestibular stimulation. Unusual stimulation, however, may effect the X nucleus, resulting in nausea and vomiting. In certain pathologic conditions all other responses may be absent, yet vomiting prove that end-organs and VIII nerve are functionating.

Not only careful qualitative observations of responses are necessary, as to the character, horizontal, vertical or rotary—and the amplitude, small or large, but quantitative determinations as well, using the stop-watch to measure the eye movements following stimulation.

By using, for the caloric tests, the temperature 68° the same in every case, and for turning tests, number of turns, rate of speed, and positions of head always the same, it is possible to standardize these tests to such a point that observations of tests repeated on the same individual give results showing no more than one second of variation, placing them well within the bounds of mathematical precision.

What practical value attaches to this new work? Last week I witnessed the rejection of otherwise perfectly adapted applicants for aviation service because of hyper-phoria, which would render probable in fatigue a false projection of the terrain in making a landing after flight, with obviously dire consequences. I also witnessed the rejection of otherwise equally desirable material for aviators because of subnormal sense of balance detected by these tests. An individual who has an impaired vestibular apparatus may be able to accomplish ordinary locomotion incidental to the course of everyday life with little recognizable inability. But put him in water whose buoyancy counteracts the effects of the pull of gravity upon which he has unconsciously been relying for much of his guidance in performing ordinary co-ordinations, and he is more or less seriously handicapped. Or let him undertake to guide an aeroplane, deprived of much of the constant information coming from his sole touch, musele-sense, joint-sense and tendon-sense, and he will be hard put to it to respond with the adequate co-ordination adjustments requisite for his maintaining himself in safe relations to his new environment. An aviator may be deprived of his visual anchor to terra firma by the intervention of cloud or darkness, but if he has the normal equipment in his vestibular apparatus, he is fully able to maintain his proper position in space. If he has an impaired vestibular apparatus (by which we mean the *entire balance mechanism*), he may still possess enough, in conjunction

with the help of his visual apparatus, to enable him to manage an aeroplane in daylight, whereas he may find that in the dark, deprived of the additional guidance of vision, he is totally unable to maintain his equilibrium in the air.

In connection with the question of sensory effects of vestibular stimulation the following narration, *in his own words,* of the experience of the aviator Miller, who while flying for France was stricken blind at a height of 7,000 feet, conveys more graphically than reams of descriptive text the subjective realization of the service of the vestibular sense in extraordinary emergency: "I was spinning along at about 7,000 feet elevation, apparently just as fit as ever in my life, when, in a flash, I went blind. The world grew dark before my eyes."

"Of course the shock was terrific, but I didn't let the idea that I was 7,000 feet away from the ground get out of my mind. And I got down safely. Experience had taught me just about how to set the planes, *and I could feel something guiding me.* My landing, they tell me, wasn't at all bad for a blind man's landing; in fact, I had hit the ground harder sometimes when I had all my faculties."

To the internist there is offered evidence of toxic nerve affections in the impairment in the normal responses to stimulation; differentiation of vertigoes, first into systematized and unsystematized types; differentiation of unsystematized into physical and neurotic varieties.

To the neurologist, the assurance that the peripheral neuron and end-organ of the vestibular part of the VIII nerve is intact or not; that the afferent neuron from medulla to cerebellar nuclei is intact or not; that the afferent neuron from cerebellum to centre for sensing motion is intact or not; that one or the other or both the two main motor efferent paths are intact or not.

To the cranial surgeon, that a lesion involving one or more of the paths has not crossed the mid-line and is possibly operable, or that it has reached proportions involving tracts from both sides and is inoperable.

The ophthalmologist finds a new direct pathway to the extra-ocular muscles. His observations of esophoria, esotropia, exophoria, exotropia and cyclophoria can be valuably supplemented by accurate observation of the contractibility of the individual muscles in response to *involuntary* as well as voluntary motor mandates.

## DISCUSSION.

**Dr. Wells P. Eagleton**, Newark, N. J., said Dr. Lewis' paper called attention once more to the great work of the Philadelphia school. After using the turning test for a considerable time he had become so confused that he discarded the chair. Dr. Jones and his associates, however, had gone about the matter in a definite and methodical manner, and their work had done much toward making the method available. One might go through a whole volume of the German Archives dealing with the subject, and yet would fail to find just the point that would make it applicable. One should not be merely an otologist, but an otolgist plus a neurologist. The vestibular apparatus furnished a means of diagnosing many conditions before the neurologist could do so, because of the fact that it gave, in many cases, the first symptoms of cerebral disease. In cerebellar pontine tumors the aural symptoms might be present for months, perhaps for years, before the neurologist could make a diagnosis on neurological signs alone.

**Dr. George Mackenzie**, Philadelphia, Pa., did not agree with the essayist's view that the slow movement after turning was an involuntary compensatory movement. Past-pointing was really a voluntary movement on the part of the patient, just as the quick component of nystagmus was voluntary. He was glad to see that his original figures for after-turning nystagmus had been substantiated. It had been his privilege, twelve years ago, to study with Barany, who stated at the time that duration of after-turning nystagmus ranged from one hundred and twenty seconds to nothing. In a paper presented before this Society two years ago he had given the reasons for Barany's failure to obtain consistent findings after turning. He had been stating his figures for the last ten years in different publications and was glad to know that the so-called "Philadelphia School" had come to accept them. There was no such thing as "after-after-nystagmus."

**Dr. Lewis**, in closing the discussion, did not believe that an otologist should be a neurologist; otology was a large enough field in itself. But it was important for the otologist to be able to refer otological cases to the neurologist for assistance in establishing scientifically accurate diagnoses. He had not employed galvanism, as recommended by Dr. Mackenzie. Inasmuch as motion was the natural stimulus of the vestibular apparatus, Dr. Lewis had limited his testing entirely to the use of turning and caloric tests in order to follow nature as strictly as possible in applying artificial stimulus for the purpose of making observations of functional responses.

## A METHOD OF MEDICATING EUSTACHIAN BOUGIES.

By LEE M. HURD, M. D., New York City

The eustachian bougie has been advantageously used for over thirty years to dilate tubal stenosis and stimulate the lining membrane.

The necessity of medicating the tube at the time of bougieing has developed several methods.

*First*: Dipping the bougie in an aqueous solution of silver or iodine.

The disadvantage of this method is that the medicament does not stick to the bougie, being rubbed off either in the catheter or at the mouth of the tube.

*Second*: Coating the bougie with a heavy base, such as lanolin. This, again, is as easily rubbed off as the aqueous solutions.

*Third*: The use of a wire bougie, such as a Holmes or a Yankauer, wrapped with cotton.

This is a much better method, but with the disadvantage that a considerable portion of the solution, into which the bougie has been dipped, is squeezed out as the bougie advances into the tube, as well as the difficulty of properly adjusting the cotton on the bougie to the size required for the particular tube in question.

The simple method, which I have used for fifteen years, has the advantage of placing the drug used where you want it and on the exact size of bougie you wish in the particular case.

Celluloid bougies are used, which are coated with silver nitrate, using gum acacia as a base.

The method of preparation.

In a narrow test tube, make a saturated solution of gum acacia in a water bath, then add the silver nitrate solution, making the solution from 1% to 10% strength of silver as desired.

The bougie is dipped about one and one half inches into this solution, and when the bougie has a uniform coating, it is placed to dry, then I usually give the bougie a second coating. When dry, the gum acacia and silver coating looks and feels like varnish to the dry fingers.

The silvered bougies can be made up in quantities, and when kept dry, will last fairly well for a week or more.

The one disadvantage of using bougies several days old is however that the silver nitrate will gradually oxidize, so that the coated bougies, a week or more old, may have lost half of their silver nitrate per cent.

I generally use them the second or third day after preparing them.

Method of introduction:

A plain gum elastic or celluloid bougie, of the proper size, is passed through the tube to make sure of the position of the catheter and the size of the bougie the tube will admit and also to dilate the tube, then the plain bougie is withdrawn, and the coated bougie can be readily passed as far as desired and remain there about two minutes to allow the moisture of the membrane to dissolve the gum and deposit the silver where desired, knowing that a definite per cent. of the silver nitrate has been deposited.

When the bougie is withdrawn, the gum coating will be entirely gone or only a little stickiness will be felt.

The pathology of the tubal mucosa is practically the same as that of the nasal pharyngeal mucosa,—acute congestion, chronic hyperplasia, chronic hypertrophy, etc.

The vast majority of tubal stenoses will readily yield to the use of the unmedicated bougie, but some cases of hyperplasia and hypertrophy are not improved by the plain bougie, then the bougie varnished with gum acacia and silver nitrate will reduce the swelling.

*Results:* One or four per cent. is usually strong enough to contract the membrane, if not, the strength can be increased up to ten per cent.

The stronger per cents should be used cautiously if it is necessary to pass the coated bougie beyond the isthmus, as several times, I have produced a serous exudate which drained into the tympanum, producing tinnitis and diminishing the hearing for several days.

I have never had pus form in the middle ear, nor had to incise the drum membrane for relief of the exudate.

The weaker silver nitrate per cents produce no discomfort, but sometimes, the stronger silver will give annoyance and cause some pain, referred to the ear, similar to the discomfort produced in the nose by 10% of silver nitrate.

Ultimate results, naturally, vary.

Some tubes remain perfectly patent, while others relapse after a time, and require a repetition of the treatment.

# THE COMPLETE MASTOID OPERATION: ITS RELATION TO THE MODERN HEALING OF MASTOID WOUNDS.

By HUGH B. BLACKWELL, M. D., New York, N. Y.

The procedure about to be described as the complete mastoid operation has been performed by the writer upon some 113 patients. My purpose in using this technique with its associated after-treatment is:

1. To prevent as much as possible the development of those serious intracranial and other complications which sometimes follow an inflammation of the mastoid or an operation for its relief.

2. To reduce the time required for the healing of the mastoid wound.

3. To render the dressing as painless as possible.

4. To improve the appearance of the healed wound.

In order to satisfy these four postulates and estimate the true value of an additional step in the technique of such an orthodox procedure as the simple mastoid operation, it is necessary (1) that the surgeon should employ this technique in a large number of operations; and (2) he should observe in a general way the post-operative course of at least an equal number of patients operated upon and treated according to other methods.

The cases upon which this paper is based are not collected ones. With few exceptions, they represent the total number of mastoidectomies performed by the author in the past few years. Practically all the operations took place at the New York Eye and Ear Infirmary or at the Manhattan Eye, Ear, and Throat Hospital.

Of these 113 patients, one developed sinus thrombosis on the seventh day of convalescence, and was successfully operated upon. In 23, well marked perisinus or epidural abscess was present; all recovered. Subperiosteal abscess was present in 18 cases. Two patients developed erysipelas during convalescence,—one a man and the other a woman,—this complication appearing on the tenth and nineteenth days respectively; streptococcus was the infective agent most commonly found, being present in 48 cases; seven were of the capsulatus variety; one patient died. One patient, an alcoholic, died of delirium tremens. Three patients of the 113 developed postoperative meningitis of otitic origin, and died. The first symptoms of this fatal complication appeared in these three

cases on the 2nd, 3rd, and 10th days of convalescence, respectively. In one of these fatal cases neither sinus nor dura was exposed, the infecting organism in this instance being the streptococcus capsulatus.

Dura covering the sinus or brain was exposed in 46 of these operations. There was a total of four deaths in these 113 cases, one of which bore no relation to the mastoid disease. In regarding this mortality, it must be remembered that these patients practically represent successive cases, many of which were brought to the hospital in advanced stages of mastoid disease. The three cases of meningitis occurred in young male adults, one of whom had a subperiosteal abscess of several weeks standing containing gas. In the other two fatal cases, the mastoiditis was produced by an encapsulated organism, one a streptococcus and the other a pneumococcus. One of these patients, prior to the operation, developed symptoms of what might have been a beginning meningitis.

The usual time required for the complete healing of the wounds varied from three to five weeks. A number of cases healed in less than this time, some requiring slightly over this period. Owing to the light packing of the mastoid wound, the dressings are much less painful than is usually the case. After the healing, the wound presents little if any deformity, the depression being scarcely noticeable.

*Operative Technique.* In making the incision for this operation, it is necessary to curve the upper extremity until it is directed horizontally forward, terminating in a point about one-eighth of an inch above and in front of the superior attachment of the auricle. (See Fig. 1.) This incision is usually carried through the lower edge of the temporal muscle along the line of the external wound. The cut in the muscle is on the bias of its fibres and is between one-eighth and one-fourth of an inch in depth. Exterior to this cut, the external surface of the tempora is freed from the auricle. The underlying periosteum is elevated. On retracting the soft parts, there is a wide and free exposure of the entire zygomatic region. These preliminaries are essential to the free exposure and curetment of the attic region. I have never found that this incision into the temporal muscle resulted in its infection or in any impairment of its function.

The bony mastoid cortex and subcortical cells are then removed, together with the tip and the cells over the knee of the sinus; the antrum is opened and curetted; the zygomatic cells are removed.

and the cap of the external semicircular canal is exposed. All gross evidence of disease having been removed from the mastoid, the operator is ready for the third stage, namely the curetment of the attic of the middle ear. (See Fig. 2.)

At this point, it may be well to review the surgical anatomy of the epitympanic region. The attic of the tympanum is that portion of the middle ear cavity which lies above the level of the tympanic membrane. It contains the head of the malleus and the greater part of the incus. These two ossicles articulate in about

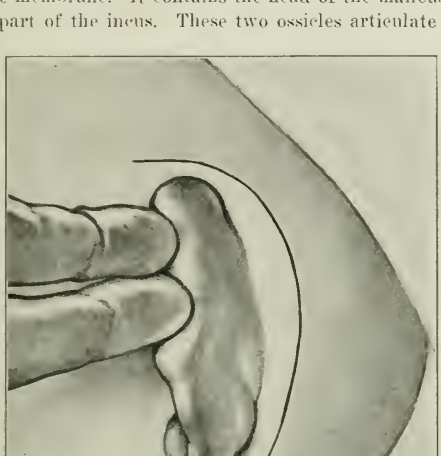

Fig. 1.—The primary incision. Its upper extremity is directed horizontally forward.

the middle of the attic. Running upward from the head of the malleus to the vault of the attic is the superior ligament of the hammer, and proceeding forward from the neck is the anterior ligament. The malleoincudal body, with the superior and anterior ligaments of the malleus divide the epitympanum, surgically, into two cavities,—the internal attic, lying internally to these structures, and the external attic, lying externally. The external attic is still further subdivided by the external lateral ligament of the

malleus into Prussak's space, which lies below, and a much larger space above, the external attic proper, with which we are now concerned.

When curetting the attic of the tympanum, the external attic should first be cleared out and the malleoincudal body exposed, after which the internal attic may be safely curetted. In curetting the external attic, a small narrow Spratt curet is passed from the antrum directly forward until the spoon rests immediately behind the bony external attic wall. The long axis of the curet should be passed parallel to the same plane as the external semicircular canal and held slightly above it (Fig. 2). By observing these precautions, we insure the entrance of the instrument

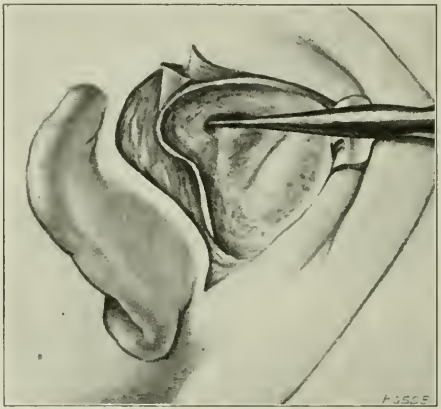

Fig. 2.—Appearance of wound at the conclusion of the simple mastoid operation. Curette in position about to remove the external attic wall. The horizontal semicircular canal is visible just below and parallel to the narrow extremity of instrument shank.

into the external and not the internal attic. The incus lies directly below and external to the spoon, and by keeping the edge of the curet in close apposition to the bony external attic wall there is no danger of disturbing the malleoincudal body lying slightly in front and internal to the back of the curet.

The external wall of the attic is then removed by curetting directly outward, the direction of motion being still in the same plane as the external semicircular canal. The posterior bony

canal wall is lowered to within about one-quarter of an inch of the membrana tympani, and is reduced in thickness. The short process of the incus should then be visible. It is commonly found embedded in granulation tissue, but is always just below and anterior to the cap of the external semicircular canal, resting in the fossa incudus (See Fig 3).

We now continue to remove the external attic wall by curetting in the aforesaid manner until the body of the incus and the head of the malleus is exposed. (Fig. 4.) In fact, in the latter stage of this portion of the operation the curet has been in contact with the anterior wall of the attic.

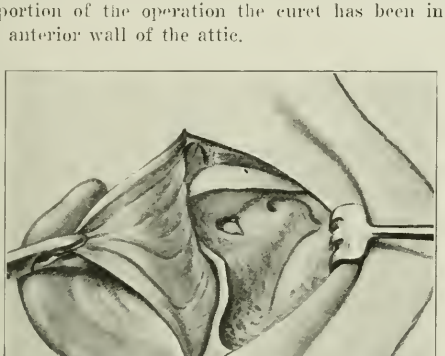

Fig. 3.—The external attic wall partially removed, revealing the body and short process of the incus lying in the "fossa incudis" just below and anterior to the prominence cap of the horizontal semicircular canal.

With the attic thoroughly exposed and the malleoincudal body freely visible, it is a simple matter to curet the internal attic. In all the cases of mastoiditis that I have operated upon, I have never failed to find the attic region filled with granulation tissue.

*Postoperative Care and Dressing.* Usually, at the conclusion of the operation, an iodoform gauze drain (from four to six inches long and one inch wide, doubled once) is laid down to the antrum region; more recently, plain sterile gauze has been employed, giving somewhat better drainage. The external auditory canal is packed snugly with a narrow strip of plain gauze

dipped in saline solution. The posterior wound is sutured from above down to within the last half or three-quarters of an inch, and a copious wet saline dressing is applied externally. This is continued for the first week or ten days, after which a dry dressing is used. Boric acid ointment or vaseline applied to the auricle and adjacent skin prevents maceration of the cutaneous surface. Daily dressings are performed until the wound has almost healed. This usually takes place within three to five weeks from the time of operation. Half of the sutures are removed on the first day after the operation, and the remainder on the second day. The canal is packed snugly at each dressing, and the posterior gauze drain is usually discontinued between

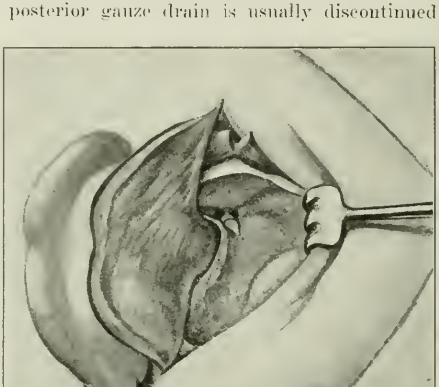

Fig. 4.—Appearance of wound at the conclusion of a complete mastoidectomy. The external attic wall has been removed, the head of the malleus with its superior ligament can be seen anterior and internal to the incus. The internal and external attic spaces lie respectively on the inner and outer sides of the malleo-incudal body.

the fourteenth and the twenty-eighth day. Owing to the small amount of packing used in the mastoid wound, the dressings are comparatively painless. After healing has taken place the amount of postaural depression is usually very slight,—in fact, hardly noticeable.

The functional results have been in every way satisfactory. So far as known, none of the patients has had to undergo operation for recurrent mastoiditis.

The writer wishes to emphasize the importance of packing the wound lightly, particularly the antrum region. Gauze packed tightly in the mastoid wound not only results in insufficient drainage therefrom but is, in the author's opinion, the most common cause of delayed healing and depressed cicatrices. This is especially true in cases where a thorough operation has been performed.

A striking feature of the postoperative course of these patients is the rapidity with which a long continued and profuse middle ear discharge ceases. The external auditory canal usually becomes dry on the second day. My own belief is that the failure to curet the attic region is frequently the cause of the persistent otorrhea which sometimes follows a simple mastoidectomy.

## Conclusions.

1. It is the writer's conviction that in order to satisfy the requirements of his four premises it is necessary to perform all mastoidectomies as thoroughly as possible.

2. He believes that the exposure and curetment of the attic region has a direct beneficial bearing on the postoperative course of these patients.

3. That light packing of the wound, using only sufficient gauze for drainage, together with daily dressings, is essential in obtaining a satisfactory result.

4. In respect to the painful dressing, slow healing, and frequent poor cosmetic appearance, the simple mastoid operation is one of the most unprogressive of modern major surgical procedures, and any technique which promises to safely modify these distressing factors of convalescence deserves the serious consideration of otologists.

## DISCUSSION.

**Dr. Norval H. Pierce,** Chicago, felt sure that Dr. Blackwell would modify this technic as he progressed in his work. He regarded it as an unnecessary destruction of a very delicate organ, the destruction not being accompanied by improvement in results which could not be obtained by very much simpler methods. He had recently operated on a child on whom, two years previously, he had found it necessary, because of the very septic course from acute otitis media purulenta, to make a thorough exposure of the tympanic antrum and to expose both sinuses. Notwithstanding the fact that both sinuses were explored, there being no external evidence of thrombus, the septic temperature went on, and the wound, therefore, was packed open, no stitches being employed. The patient recovered, fortunately, and the result was a much depressed scar; and, during the two years, there have been three recurrences of otitis media: at each attack the scar would swell, the

soft tissues would become inflamed, and the collection of pus in the mastoid could be squeezed out through the perforation in the tympanic membrane. Finally it was decided to open up and see what had taken place. The attic and tympanic antrum communicated with a fistula which extended backward and downward, very near to the knee of the sinus. The bone had completely formed over the surface, with the exception of a very small area (about one and a half inches) over the bone posteriorly. The bone below had formed, but there was a sac from the antrum to the posterior extremity of this cavity. He had not received the histological report on it yet, but he felt sure the sac was caused by the growth of the mucosa from the epitympanic space into this space. In other words, the mucosa had spread more quickly than the bone cells could form bone, and therefore a permanently enlarged antrum was formed, lined by mucosa of low vitality, which became inflamed every time the ear became inflamed. This was not an isolated case; he had seen many similar cases. From these experiences he was convinced that it was not good practice, where it could be avoided, to open the antrum thoroughly,—posteriorly and anteriorly,—and curet it, because of the danger of the spread of the mucosa over the hard interior table of the skull, which is poor in blood vessels. It was better to drain from the bottom. He did not advocate in any case, leaving diseased bone around the antrum; but there was diseased bone around the antrum in very few cases. He was yet to be convinced that the hearing was not impaired in the cases operated on in the manner advocated by the writer. The mechanism involved was so finely formed that it could respond to vibrations so fine that they are beyond the power of our microscopes to measure, and it was difficult to understand how this could be interfered without impairing its function.

**Dr. Joseph C. Beck**, Chicago, Ill., agreed with the views expressed by Dr. Pierce. He asked the essayist to definitely state his premises, as he had found conditions on the operating table. During the last ten years he had not gone beyond the antrum in the first part of the simple mastoid operation, conservatively avoiding disturbing the lining membrane of the antrum. Experience had demonstrated that healing was much more rapid, and that the function was better, in acute cases, when the antrum was left alone. If, however, Dr. Blackwell had reference to chronic cases that would not recover from the simple operation, the procedure would be welcomed.

**Dr. William B. Chamberlin**, Cleveland, Ohio, was strongly of the opinion that a paper which advised an operation of such extent in simple mastoid cases ought not to be presented before this Society without challenge. Challenge would doubtless not change the essayist's opinion on the subject, but the Society should not go on record as sanctioning a procedure of this character. The operation described was little more or less than the Heath operation, with the exception that the posterior canal wall was not removed. He had learned from sad experience that any disturbance of the ossicular chain would lead to marked impairment of hearing. Neither Dr. Blackwell nor anyone else could enter the attic, as described, without disturbing the ossicular chain. In a case in which he had inadvertently dislocated the incus, the effect on the hearing was pronounced. The patient's hearing was markedly impaired, and always would be. As to the ear being dry in two days, he could show case after case in which the mastoid operation had been performed, where the canal was free from discharge at the first dressing. The only point on which the speaker agreed with the essayist was with reference to packing. Long experience had convinced him that in many cases convalescence had been unduly protracted by zeal in packing. In cases referred to him by others he had pulled the packing out, left the wound wide open, and healing had quickly taken place.

**Dr. George L. Richards**, Fall River, Mass., referred to the excellent work of Dr. Beck in connection with surgical pathology and repair of wounds. How would these large wounds heal? The cases looked beautiful when finished, but what about the healing? The cases which healed best and most quickly were those in which nature had prepared things for the surgeon.

**Dr. Charles W. Richardson**, Washington, D. C., emphasized the point that while this operation, as detailed by Dr. Blackwell, might be well done by him, it was a dangerous thing to go out to the average man practicing otology and performing mastoid operations. The end results, in at least seventy-five or eighty per cent. of the cases, would be functional impairment from dislocation or injury to the mechanism of the malleo-incudal joint. If Dr. Chamberlain could report a case in which he removed the incus, there was very great liklihood of the less expert doing the same thing. The majority of surgeons performed the mastoid operation in the same manner, as it had been learned from experience and judgment that too much meddling in the antrum and attic was bad for the patient. Recovery was not so rapid, and granulation seemed to be arrested more frequently than where less meddlesome work was done.

**Dr. Blackwell**, in closing the discussion, said that the indications for performing the "complete mastoid operation" and applying its associated after-treatment were stated in the premise of his paper.

He believed that among formal major surgical procedures the mastoid operation was one of the most unprogressive as regards the modification of its attendant distressing postoperative factors and for the reason suggested in his paper there were few major operations which the average physician or layman so dreaded as a simple mastoidectomy. He, for one, did not consider that the last had been written or said concerning the proper surgical technique of even so formal a procedure as the simple mastoid operation. Furthermore, he believed, if as suggested in his paper the gentlemen who were unfavorably impressed by this technique would only employ it in operating upon a large number of cases and compare the results obtained with an equal number of similar cases, operated and treated according to other methods, they would soon arrive at some definite idea of its value. Dr. Blackwell said that he had read Mr. Heath's account of his operation and could find but little analogy between the two operations. In the first place, Mr. Heath did not curet the attic of the tympanum and, in fact, advised against it. He only curetted the antrum. In addition, Mr. Heath closed the posterior wound, cut a plastic meatal flap, and dressed the wound per canal. For the benefit of the gentleman who had made the comparison, he would say that he had performed the complete operation with some modifications on a series of chronic otorrhoeas. This paper was read at the meeting of the American Otological Society held May, 1916.

# REPORT OF A CASE OF SIMPLE MASTOIDITIS COMPLICATED BY PARATYPHOID FEVER WITH METASTATIC ABSCESSES.

By DUNCAN MacPHERSON, M. D., New York, N. Y.

The liberty of asking for a few minutes of your time to hear the report of this case is taken for the following reasons:

1. Because paratyphoid complicating mastoiditis is rarely mentioned in the literature.

2. The advisability of drawing the attention of the otologist to its possible presence, inasmuch as it may insidiously disorder our more frequent pictures of ear disease, causing confusion and embarrassment in diagnosis unless laboratory help is freely drawn upon.

3. Mastoiditis with acute onset; very little bony destruction, negative culture at the time of operation, but followed by remote abscess formation.

4. The temperature, suggesting a possible irregular manifestation of lateral sinus thrombosis.

5. The laboratory help.

(a) By negative blood cultures except for the finding of paratyphoid "a."

(b) The blood count confusion with pus formation present.

(c) Negative Widal and diazo reaction.

(d) Streptococcus haemolyticus found in the pus taken from the abscesses.

(e) The finding of paratyphoid "a" in the blood.

In the scanty literature where a paratyphoid bacillus is referred to in mastoiditis, the type "b" is found responsible.

Of the six (6) cases found reported and bacterially differentiated, two are believed by the authors to have been primary in the mastoid. In one, where general indisposition preceded the paratyphoid mastoiditis, the belief is expressed that paratyphoid fever was overlooked, influenza being held to be responsible for the paratyphoid symptoms,—no intestinal disturbance having manifested itself. In one of the cases it is noteworthy that the patient was admitted to the clinic suffering from sore throat, a condition that is known to occur in typhoid fever. By way of the pharynx,

the infection could have been carried to the middle ear. Anatomically, two cases were characterized by a peculiar hyperemia of the bone, presenting an appearance different from that ordinarily seen in other suppurations.

In one instance, the authors report the findings in the case of a child who died of melana neonatorum, 36 hours after birth. Both ears contained pus. The paratyphoid bacillus "b" was found in the cadaveric blood and also in the pus from the ear. The mother had had an attack of paratyphoid four weeks prior to the birth of the child. In another instance, a patient eight weeks old, the author observed paratyphoid meningitis complicated by severe gastro-enteritis. The autopsy showed, in addition to these diseases, a double otitis media, and cultures of the paratyphoid bacillus "b" grew from all the organs of the body.

In 1914-15, a report from the French hospitals refers to an epidemic of typhoid complicated in ten per cent. of the cases by suppurative otitis, often of a severe type. Many cases of paratyphoid were found, especially after the subsidence of the typhoid epidemic. Otitic complications were also frequent in the paratyphoid cases, but not of so severe a type as in the typhoid. The onset was insidious, with low temperature and no pain, an aural examination being necessary to demonstrate the trouble. No reference is made in this report as to whether the infection was of type "a" or "b."

In cases reported from Italy and others elsewhere from the Western front, no differentiation was made as to the type of paratyphoid infection.

The patient called at my office on the evening of April 17, 1915, saying that her ear felt queer, that some years before she had had abscesses in her ear, and that the feeling simulated that present on the former occasions.

Examination did not reveal the slightest sign of acute inflammation. The middle ear was gently inflated by means of the catheter, and the tube seemed to be wide open.

The following morning, at about ten o'clock, she returned, complaining of an increase of the discomfort. Examination showed signs of slight tubotympanic involvement. Dry heat was prescribed, supplemented by hot douches should pain make its appearance and was not relieved by the dry application. Directions with reference to diet and general elimination were given.

At 11:30 I was requested to call at the patient's home, as the

pain was said to be intense. Arriving at 12:00, two hours after finding the tubotympanic involvement at the office, I found marked bulging and redness of the entire drum. A free incision was made. No culture was examined. During the next five or six days the temperature oscillated from normal to 102° by mouth. On the seventh day following the incision, increased mastoid tenderness and some edema of the posterior superior wall was present, and another opening was made, carrying the incision deeply through the prolapsing canal wall. The temperature rose to 104.5° and the following day,—the canal showing considerable narrowing and the patient complaining of marked tenderness on slight pressure, especially over the mastoid tip and antrum,—an exploratory operation was decided upon.

Owing to the acute onset, the marked diffuse tenderness, the bulging canal, and the amount of discharge, I expected to find considerable involvement, but there was only slight softening of the cells around the antrum. The tip cells were explored.

The next day, at 7:30 p. m., the temperature rose to 105.2°, with a pulse of 102 and respiration 22. The patient was comfortable, but had a chilly feeling, followed by perspiration and accompanied by some headache. An ice bag was applied to the head, an alcohol sponge was given, and five grains of aspirin were prescribed by Dr. Brugman, the consultant.

The following morning the temperature was 102°, but it gradually rose until at 9:00 p. m. it was 105.6°. A chilly feeling preceded the rise and sweat followed. With alcohol sponging, the temperature fell to 103.4° at midnight. The patient had a comfortable night and did not seem to be as sick as the symptoms would lead one to expect. This relatively comfortable feeling persisted throughout the attack.

The eye-grounds were examined by Dr. Dwyer the day following the operation and were found to be normal. A differential blood count made on the day following the operation showed: Leucocytes 10,800; polynuclears, 73%; small lymphocytes 22, large 5.

Five blood cultures were made from April 28th on. Four were negative; the fifth showed paratyphoid "a". The culture from the blood was tried out in all the ordinary media and corresponded in every way with paratyphoid "a".

Widal on May 1st was negative, and though repeatedly looked for throughout the disease, continued absent.

The urine showed no diazo reaction throughout. Catheterized

specimen showed staphylococcus. No malarial plasmodia were found at any time.

The pulse and respiration remained almost the same throughout, i. e., 76, 120 and 18-24.

The wound was clean throughout but granulated slowly.

On April 30 the patient complained of her daily chill, headache, and perspiring period, and of a pain in the region of the left hip and lower abdomen. The temperature rose to 105.6° and fell in the evening to 100°, rising the following day after a chill to 105°. The patient slept well, was cheerful, and had less pain.

This oscillating state of affairs continued to May 9th, when the rectal temperature fell to 99.4°. The hip pain was much less.

On the 11th there was another chill and the temperature rose to 105.8°. Throughout the attack the highest leucocyte count was 10,800; the polynuclear 73%, and small lymphocytes 26%. Blood cultures were negative except for paratyphoid "a". The hip continued to feel stiff and the pain, while present, varied in degree, at no time being intense. Sore throat and hoarseness were complained of, but no sign of laryngeal cartilage involvement was found.

On the 30th, the temperature rose to 105.8°, falling on the following day to 100.8°, from which date to June 17, it oscillated between normal and 103.4°,—the patient complaining very little of her hip. On June 17, the hip abscess was opened by Dr. Brugman, and the temperature fell to normal on the 19th, and so remained until June 26th, when it suddenly rose to 106°, following the opening of a scalp abscess posterior to the mastoid wound. The wound itself was clean and the abscess did not communicate with it. The temperature fell to 101° and the following day rose to 106°. A blood culture was made,—looking again for a possible sinus involvement,—but proved to be negative. The temperature then gradually fell to normal. On July 1st, it rose to 102.6°, following the opening of a small scalp abscess in the region of the parietal eminence. The temperature then fell to normal, rose to 102° on July 5th, when another small pocket of pus was found midway between the mastoid and the eminence. This was opened and the temperature fell to normal on July 6th, and did not rise again. Streptococcus haemolyticus was the bacterial finding from the abscesses.

To what extent the paratyphoid alone would have altered the picture of an otherwise healthy postoperative mastoid, I am not

prepared to say. What effect the paratyphoid played in the formation of the abscesses, I am not prepared to say.

The normal blood counts may be suggestive of the effect of paratyphoid "a" on blood differentials in the presence of pus, although normal blood counts are found in other acute conditions where pus is present in the body, and bacteremia may escape detection even in lateral sinus thrombosis as pointed out by Dr. Dwyer, unless the blood is cultured frequently and with special reference to the time of the chill.

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# REPORT OF INTERESTING NASAL CASES PROBABLY DUE TO SYPHILIS. REMARKS ON OBSCURE SYPHILITIC NASAL SYMPTOMS.

By DUNBAR ROY, M. D., Atlanta, Ga.

Obscure syphilitic manifestations in the nasal cavities are much more frequent than many of us suppose. Since the introduction of the Wassermann test the writer believes that we have made ourselves too dependent upon it as the final test in diagnosis, to the exclusion of clinical symptoms which are so important in the management of pathologic conditions. The following case is presented in detail as affording many points of interest to the neurologist, the internist as well as to the rhinolaryngologist.

## CASE REPORT.

Mr. E. E., aged 51, cotton merchant. Patient had always been strong and healthy, never having had a severe spell of illness during his life. Is temperate in both drinking and smoking, in fact seldom takes a drink. Has lived in Atlanta most of his life. His father and mother are both living, and both in excellent health. In Aug., 1902, he moved to Shreveport, La., to represent a northern firm as their cotton buyer. In that city he seemed to enjoy good health, remaining at his usual weight of 150 pounds. Patient was not over four feet nine inches in height. Had spent each summer in Atlanta, returning to Shreveport in the fall. During the winter of 1903-04 he did not feel in his accustomed good health, and along in the spring of 1904 began to have neuralgia and drawing sensation in the muscles in the back of his neck. In May, 1904, he returned to Atlanta to live, having made new connections in the cotton business. On arriving in this city he consulted his physician on account of this severe neuralgic pain in the back of his neck. The appetite was not good and he was already beginning to lose flesh. Being a neighbor and a warm personal friend of mine, I saw him quite frequently but in an entirely unprofessional manner. In this way I noted his gradual decline. His physician attributed the trouble to muscular neuralgia, as a result of malarial intoxication. He was now treated steadily but without improvement, while the pain gradually extended down into his shoulders and arms. These pains grew to be excruciating and would come upon him in paroxysms. His digestion and appetite were poor, and his nightly sleep very much disturbed. Not improving (which is characteristic of a great many patients, and we can hardly blame them) he called in another physician, who took charge of his case. He also attributed the cause to malaria, and immediately began to dose him with various medicines, changing the same from time to time. He was put upon a strict diet, and his liver was kept very active. Soon a derangement of the function of this organ was diagnosed as playing a prominent role in producing the various physical ailments. The patient was blessed with an excellent nurse in the shape of his wife, who rubbed and massaged him with various liniments and looked after his every physical and mental comfort.

The pain, however, did not subside, but was extending still further down the patient's back, and even into his legs. At no time were there any signs of paralysis, and the patient was able once a day to go to his office and remain for a few hours.

In giving the history the patient told me that his physician made a thorough examination of his secretions, urine, etc., and found nothing abnormal. The pains in the shoulders, especially the left, and in the back were sometimes accompanied by such spasms of the muscles as almost to draw the head to the shoulder. In September, five months later, the patient, not improving but seemingly growing worse, becoming weaker each day, losing flesh, and the pains being so severe as to render sleep impossible except for a few hours at night by means of codein, he consulted a renowned osteopath, who told him it was due to the nerves from the spinal column. He gave him three treatments a week and put him upon a still more restricted diet. This treatment he continued for a month, having discarded all medicines except certain mineral waters suggested by friends. At the end of this time he was no better in any respect, the pain not having been relieved, they being now in the legs as well as in the arms, and accompanied by the most violent spasms. At this point the patient began to be hoarse, but without cough or expectoration.

During a social visit he remarked that he had taken a severe cold, and if he did not get better was coming to me professionally. In the meantime he continued to have thorough bodily massage but being dependent upon medicine to bring sleep. It was frequently remarked among his friends that Mr. E. looked like a doomed man and they believed that he would live but a few weeks. On my return to the city after the Christmas holidays, his wife telephoned that Mr. E. wished to consult me in reference to the severe cold in his head and throat. Being a personal friend and a neighbor, I was really sorry that the patient had come to me for professional advice and treatment, for with his previous history in regard to his medical treatment (of which I was cognizant) I felt that I could do but little for him now that he seemed in the last stages of some obscure nervous disease.

When he came into my consultation room he was so weak, feeble and emaciated that I felt as if he would never be able to return to his home. His weight then was 95 pounds, having been reduced from 150. He had no signs of paralysis, his reflexes were normal, and his only complaint being the intense pain and spasms in the legs and arms accompanied with the gradually increased weakness.

Two weeks previous, according to statement, he contracted in addition to the hoarseness, a cold in the head, and this had gradually kept up until at present it was quite disagreeable, having hardly sufficient strength to blow the tough secretions from his nasal cavities. His throat also, i. e. the larynx, worried him considerably by being so tight as to interfere with his breathing, especially at night.

*Examination of nasal cavities.* At the muco-cutaneous surface in both nostrils, the parts were thickened and inflamed from irritation produced by the nasal discharge. The right cavity seemed pretty well filled with muco-purulent secretion. After shrinking the tissues with cocain and adrenalin and by assistance from both patient and myself, the cavity was fairly well cleaned. Far back on both septum and middle turbinal there was a dirty, yellowish membrane covering the parts which extended also into the naso-

pharynx. A slight manifestation of the same condition was found on the left side but not quite so marked. The parts bled when some of the membrane was detached. By means of cotton on the end of an applicator I detected roughened and exposed bone high upon the septum at its posterior free border and also the same condition at the posterior end of the middle turbinal on the same side, i. e. the right. None was detected on the left. The pharynx showed nothing abnormal, but there was a large amount of tough yellowish mucopurulent secretion coming down from the nasopharynx and clinging to the postpharyngeal walls. It was impossible to inspect the nasopharynx on account of the relaxed condition of the palate and the gagging produced in my efforts to use a palate retractor.

On inspecting the larynx, I found a beefy-red swollen condition of the whole laryngeal mucous membrane, the cords being of the same general color. The membrane there and further down in the trachea seemed to be producing a tough, membranous secretion which it was impossible for the patient to expel. It gave the sound of a "croupy cough." He had no pain either in his throat or nose, but complained some of a pain just at the base of the nose over both frontal sinuses. Transillumination of these sinuses showed no shadow at any point.

The objective picture here presented was to me quite significant of a specific condition, but close inquiry elicited not one ray of such a history. I am sure the patient would gladly have told me if such had been the case, knowing that the treatment then would be much more effective. I knew him intimately and there were few men who lived a life of more sobriety. Even before his marriage he was never given to the slightest dissipation. The only other condition which such symptoms were likely to represent was tuberculosis, and this to me almost seemed the correct diagnosis. I had the secretion from the nose and throat examined but this was negative for tubercle bacilli. The loss of weight, hoarseness, slight cough, were symptoms quite significant, especially after the negative specific history. However, I knew that histories were often uncertain, and as an experiment in conjunction with the local treatment of cleansing and nitrate of silver applications, I put the patient on the following treatment: On account of the poor appetite and atony of the gastric function, he was given ten drops of equal parts of tincture of nux vomica and compound tinct. of gentian in a little water and corn whiskey before meals. After eating meals he was started on five drops of a saturated solution iodide potash in one-half glass water, and this was increased one drop every day. On the morning and afternoon he was given an expectorant mixture of carbonate of ammonia, fluid ext. of squills, etc., this being used to soften and aid the expulsion of the tracheal and laryngeal secretion. He was not limited to his diet, but told to eat as much nutritious food as possible. He was also to use a douche of warm saline solution in the nasal cavities three or four times daily.

The next day being very cold patient did not report, but his wife telephoned me that a swelling had developed just at the root of his nose accompanied by some pain on pressure. In the evening I called and found the patient sitting up in a big chair with a swelling, puffy and globular looking, about the size of a walnut, just at the root of the nose. It had developed slowly during the day. I advised hot fomentations every hour—a good saline internally, and the other treatment to be continued as prescribed. The next day when seen the edema or swelling had extended to the lids of both eyes until they could not

be opened and even down on the cheek and nose. The original point of the swelling was beginning to subside. The day following Dr. Block, a neurologist, was called by me in consultation. Dr. Block made a very thorough physical examination of his heart and lungs, muscles, nervous reflexes, and urinary secretions. This latter was found to contain a tract of albumin but no casts.

Dr. Block was unable to suggest anything further except to give the patient a mixture containing the bromides, to be taken at night for his extreme nervousness. Edema remained limited to the face, and this gradually disappeared entirely in one week.

From this time on the patient began to improve and the internal remedies were never changed, the iodide being increased to fifty drops three times daily. I saw him at my office every other day, cleansed thoroughly the nasal cavity, touched the necrosed condition in the nose and nasopharynx with a strong solution of nitrate of silver, followed finally with a covering of aristol powder. Inhalations of terebene in benzoinated albolene were given for the larynx and trachea. One morning about three weeks after the beginning of the treatment, the patient blew from his nose a mass of secretion and a small piece of bone which proved to be a part of the vomer.

The patient was treated at gradually varying intervals until August first of 1905, i. e. seven months, when he was dismissed apparently cured except that he was still hoarse from the hypertrophied condition of the laryngeal mucous membrane. I tried astringents and sprays, but this seemed to do but very little good. The medicine was never changed during the whole course of treatment, the remedies being gradually left off with the improvement of the patient's condition.

December 28, 1905, one year after I first saw him professionally, he weighed 150 pounds, and with the exception of slight hoarseness he is as well and as strong as at any time during his life. There is no discharge in either the nasal cavities or the nasopharynx. Up to the present time there has been no further trouble. He is still in the best of health now for 14 years.

*Remarks.* This case presents many interesting as well as obscure features. In the beginning of the patient's trouble, there were some symptoms of a malarial neuritis, and in fact all the physicians consulted during the first months of his illness attributed the cause of his indisposition to malaria. The very first symptom noted by the patient was a general feeling of malaise and loss of appetite. This occurred during the last months of his residence in Shreveport. In May, 1904, he returned to Atlanta weakened in health and suffering with drawing pains in the muscles at the back of his neck. There was never any rise in the bodily temperature throughout the course of the disease, nor was there at any time signs of paresis in any portion of the body. It is just possible that a slow grade multiple neuritis might produce some of the symptoms in this case, but my own observation and that of other observers would not lead us to such a conclusion. For indeed at no time during the illness were there signs of paresis, but most

prominent were the signs of spasms and intense neuralgia of the sensory nerves.

The diagnosis of malarial intoxication was certainly a most natural one, and it is still a question in my mind whether the beginning trouble could not have been attributed to this cause.

The brilliant result obtained by the internal administration of the iodide of potash almost compels one to acknowledge the specific basis of the whole constitutional derangement, and yet, with the exception of this one fact, the good results obtained by the internal administration of the iodides, there was absolutely no history or symptoms to warrant us in drawing such a conclusion. I examined him thoroughly at the time, and even since then there has not been the slightest signs of glandular enlargement of any portion of the body. No falling out of the hair, no symptoms of sore throat, no obscure pains at night, absolutely nothing to warrant us in suspecting syphilis as the cause. So foreign to my mind was syphilis as the cause of the naso-pharyngeal ulceration at the time of these very active symptoms, that I concluded the ulceration was without doubt tubercular, coupled with the great loss in weight and the seeming involvement of the bronchial tubes. However, Dr. Smith, an excellent pathologist, made a thorough examination of the secretions with absolutely negative results. His mother had been treated for years for a superficial skin cancer on the forehead, and this is still under the constant observation of a dermatologist.

Another symptom which was misleading to me was the existence of an odor from the nose in no wise characteristic of that experienced in the case of late tertiary syphilis where the odor is exceedingly offensive, and especially where there are areas of necrosed bone. While many things point to the correctness of our diagnosis, we must remember that the administration of the iodide of potash often proves of benefit and even curative in cases other than syphilis, as for instance an alternative in various conditions where we can give no better name than "blood dyscrasia" this remedy is often of marked value. As an adjunct to the relief of various inflammatory exudates in the body, I know of no one remedy that is of greater value.

Consequently, because the case responded to the iodide of potash and seemingly was brought to a successful issue through such administration, is no self-evident proof that we were correct in our diagnosis.

The question could very naturally arise, was malarial intoxication the primal cause of this physical derangement and did its presence in the system so impoverish the blood and thereby the nerve tone as to produce the train of symptoms which occurred throughout the course of the disease? In my own mind there is such a possibility, and yet in studying the literature of any similar case reported I can find nothing which would lead me to such an affirmative answer. After all that can be said, the history of this case and the results obtained by the treatment certainly bear out the probable diagnosis of its specific character.

Another case evidently specific in character, occurring in my practice several years ago, had many obscure points in its diagnostic history, and yet the final results must lead us to the same conclusion as was obtained in the first case.

The second case is that of a Mrs. M., age 55, widow. This patient was a woman of most excellent standing. She was the mother of four children, all of whom were unusually robust in their physical growth. The family history precluded any idea of specific taint. The father and husband died several years ago of typhoid fever. The patient had always enjoyed the best of health and had never suffered from any of the feminine complaints.

*Present history.* In April, 1903, patient consulted me on account of a cold in the head which had been present for two weeks and which did not improve after the use of all the ordinary remedies. Her chief complaint was a stuffiness and inability to breathe comfortably through the nasal cavities. There had been some symptoms of tenderness about the external part of the nose whenever the handkerchief was used. With this exception her general health was perfect and there had been no signs of constitutional derangement.

Examination showed some swelling of the whole outside of the nose giving her the appearance of the so-called "frog-face." Nasal respiration was difficult, producing the whistling sounds so characteristic of an intranasal swelling. On examination with reflected light the anterior part of the septum was thickened and edematous resembling very much the appearances seen in an abscess of the septum. This could not be reduced to any appreciable extent by the use of cocain and adrenalin. The outer walls including the turbinates were only slightly swollen. There was no history of traumatism. This was before the time of the Wassermann test and hence this could not be used. The question of malignancy could very readily be suggested. There was no bleeding when the septum was touched and only the thick leathery sensation was imparted to the nasal applicator. There was very little space left between the septum and the outer walls. The pharynx and nasopharynx were absolutely normal in appearance. No glandular enlargements could be elicited.

However the latter seemed to me the most probable diagnosis from all the clinical features of the case.

*Treatment and results.* The patient was placed upon rapidly increasing doses of the iodide of potassium. The fact that this medicine was tolerated with the greatest ease indicated somewhat that the condition would respond to this line of treatment. Almost immediately the parts responded and in a few

days the patient began to loose the stuffy feeling in the nose and respiration became much easier. No other treatment was used with the exception of an oil spray which was only for comfort.

The treatment was continued for three months, at the end of which time the nasal cavities were perfectly normal in appearance and up to this time, 13 years, there has been absolutely no return of this or any other condition resembling a complication of specific dyscrasia.

*Remarks.* Of the certainty of diagnosis there of course may be some difference of opinion, but the results obtained by the use of the iodide of potassium would rather indicate the specific nature of the affection. As was stated at the beginning of this paper, the writer believes that there are many obscure lesions in the nasal cavities which have their dependence upon syphilis. It is not always necessary to look for ulcerations and even destructive processes in order to have a picture of nasal syphilis. Manifestations of specific lesions in the nose resemble many other affections, and the fact that we sometimes get such poor results in intranasal operations may have its real cause in a failure to recognize a constitutional systemic dyscrasia which in one word is syphilis. The rhinologist who waits for typical text-book pictures of lesions or is entirely dependent upon clinical tests will fail in many instances to bring about a successful result. The obscure ramifications of the syphilitic virus are manifold in character, and we can never be too diligent in instituting anti-specific treatment if no other cause for the condition can be found.

Congestion and edema of the nasal mucous membrane, as is sometimes found in the early stages of syphilis, very frequently resembles those cases of vasomotor rhinitis in all its manifold symptoms.

A typical case of this kind occured in my practice three years ago. Mrs. E. E. L., age 44, consulted me in Feb., 1915, with what she denominated a severe cold in the head. Patient was confined to her bed complaining chiefly of the inability to breathe through the nose, frontal headache, sneezing, all the symptoms of acute rhinitis. Patient said she had been having periodic attacks of this kind for the last year.

Examination showed congested and edematous turbinates with a serus discharge. Under local applications of cocain and adrenalin the membrane was with difficulty contracted. The writer has always found such a condition characteristic of vasomotor rhinitis and syphilitic congestion. Ordinary rhinitis will show contraction immediately under cocain applications thus differing from the two former conditions. The usual treatment was instituted but with slow and imperfect results. The patient was under the writer's care for several weeks and it was only by continuous treatment that she was made even partially comfortable. The greatest complaint being the stenosed condition of the nasal cavities at night. This was relieved during the day especially when the patient was out in the open. A symptom still further characteristic of

vasomotor rhinitis. It seemed as if this latter condition was the proper diagnosis. However the patient's progress was exceedingly slow and there were repeated exacerbations of all the nasal symptoms. An examination made two months after the first observation of the patient revealed a small superficial ulcer on the septum opposite the middle turbinate on the left side. Syphilis had not been suspected on account of the excellent social standing of the patient. A Wassermann was immediately made which was returned plus three positive. This patient was immediately placed upon rapidly increasing doses of the iodide potassium with a corresponding improvement of her condition. The ulcer healed immediately and complete relief from all the symptoms of nasal stenosis rapidly disappeared together with the accompanying vasomotor disturbances.

It is now three years since this attack and the patient has had no further trouble.

In conclusion, the writer cannot urge too strongly the importance of the syphilitic virus as the fundamental cause of many obscure rhinological conditions which resist the ordinary treatment. Nor should we be governed entirely by such tests as the Wassermann in directing us to the proper line of treatment. During the last twenty years the writer as the head of a large free clinic where the majority of patients are negroes and where he has seen syphilitic lesions of every known variety, he has adhered entirely to the old treatment of iodide of potassium and mercury and with few exceptions the results have been entirely satisfactory if this treatment is given from year to year.

## DISCUSSION.

**Dr. Charles W. Richardson**, Washington, D. C., suggested the wisdom, in such cases, of eliminating all thought of the social position of the patient. Any human being was subject to invasion by syphilis. Acquired syphilis among clergymen and congenital syphilis in the offspring of clergymen was not unknown, as his own case records showed.

**Dr. Lewis A. Coffin**, New York City, had frequently found the voice a very reliable indication of the presence of syphilis involving the nasal structures. There was a peculiar nasal sound in the voice under these circumstances. He could recall several cases in which the diagnosis of syphilis had been made from the voice, in one instance before he had seen the patient, and laboratory tests had substantiated this diagnosis in every case.

**Dr. Joseph H. Abraham**, New York City, agreed with the essayists regarding the frequency of this affection. He was reminded of what Chiari, of Vienna, had said some years ago, in presenting cases of obscure lesions of the upper respiratory tract: "When in doubt, treat the patient for syphilis." About ninety per cent. of cases of this category were syphilitic conditions. He refrained from using the douche in such cases. If salvarsan were administered for syphilis involving the antrum, septum, and neighboring structures, care should be taken with regard to the dose employed, beginning with a small dose, about 0.2.

**Dr. J. W. Jervey,** Greenville, S. C., quoted, in this connection, the epigrammatic declaration of a great teacher: "Know syphilis in all its manifestations, and what remains to be learned will not stretch the piamater of a megalocephalic senior medical student."

**Dr. Walter B. Johnson,** Paterson, N. J., thought this case suggested the need for coordination in medical work. The axiom, "Treat them for syphilis," seemed to apply in other specialties, but had not been appropriated by some who treat this particular class of cases.

**Dr. Roy,** in closing the discussion, reiterated that there were many manifestations of syphilis in the nose which were not recognized, and for that reason the cases did not recover. He cited a case of a woman who had vasomotor rhinitis and a corrugated condition of the nasal mucous membrane. He put her on iodide of potash and it cleared up. Another physician had recommended submucous resection. The corrugated appearance of the mucous membrane indicated a constitutional dyscrasia. In many instances the condition was obscure so far as treatment was concerned, but further investigation would reveal the specific basis.

# THIRD YEAR OBSERVATIONS OF CLINICAL OZENA AND ITS VACCINE TREATMENT.

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## Introduction.

In the past year our investigations relative to fetid ozena have been chiefly clinical. Our work has been considerably enhanced by virtue of the prize award of the Pacific Coast Oto-Ophthalmological Society, whereby we were enabled to procure suitable cagings for the animals used in these studies. We have enjoyed, also, the co-operation of many rhinologists throughout the country and take this means of thanking them for the submission of material for bacteriologic study, for clinical data of cases under observation, and for many valuable suggestions pertaining to our work. In order to acquire a broader expression of the clinical aspects of atrophic rhinitis, and to determine what general value has been attained through vaccine therapy in this condition, a comprehensive questionaire has been submitted to all interested in this subject.

In our previous bacteriologic studies we were impressed by the striking resemblance of the coccobacillus foetidus ozenae of Perez to B. bronchosepticus, the specific organism of canine distemper. To facilitate the bacteriologic nomenclature we had deemed it advisable to group the coccobacillus of Perez with B. bronchosepticus and proposed its designation as B. rhinosepticus as a more fitting appelation. This change in name we have followed in our recent contributions upon this subject.

It was our purpose to limit our investigations along certain specific lines and to determine:—

1. The existence of different types of ozena upon a bacteriologic basis, and to establish the relationship of atrophic rhinitis to accessory sinus disease.

2. The clinical relationship of B. rhinosepticus and the B. Friedlander group to ozena.

3. The value of specific vaccine therapy in the various types of atrophic rhinitis.

We have tabulated these results and note a considerable degree of similarity in expressions of the clinical aspects of ozena as well as results attained through vaccine administration. The brevity of questionaire answers can not fully allow individual expression and we are therefore incorporating in this report such personal communications as are pertinent and more illuminating to the subject in hand.

### Clinical Differentiation of Types.

It appears apparent that the inclination to group all types of atrophic rhinitis with crusting and odor as fetid ozena, and to consider them from the same etiologic viewpoint, or apply to them the same therapeutic measures, is obviously incorrect. In our previous communication on this subject we laid stress upon accessory sinus involvement as complicating fetid atrophic rhinitis, and presented such bacteriologic studies to demonstrate the constant presence of B. rhinosepticus or some type of the B. mucosus capsulatus group in all cases of fetid atrophic rhinitis. We are convinced that in these cases the accessory sinuses are too frequently disregarded, and that turbinal atrophy is at times a secondary process. Mechanical intranasal distortion, allowing accumulation and organization of secretions, might create a favorable environment for bacterial lodgement, especially for the Friedlander organisms. Permanent escape of the accessory sinuses from infection from a chronically infected contiguous membrane, seems hardly possible. In more than fifty per cent. of cases of fetid atrophic rhinitis under study there was definite accessory sinus involvement, especially of the sphenoidal and ethmoidal cells. Whatever the nasal pathology may be, its importance seems distinctly subordinate to the condition of the contributal nasal chambers, and demands a different consideration from fetid atrophic rhinitis simplex.

Whilst a certain conflict of bacterial entities, namely B. rhinosepticus and the Friedlander group organisms, still prevails in culture findings, the constant presence of one of these types in all cases of true atrophic rhinitis (fetidus) lends encouragement to the establishment of a bacterial cause.

There is still a third group, presenting some odor, a thickened dirty mucoid secretion rather than crusting, and turbinate hypertrophy rather than atrophy. Sinus involvement may or may not

coexist. The organism rhinosepticus is never recovered from these cases although the Friedlander bacillus is not infrequently isolated in luxuriance. There appears to be neither clinical nor bacteriological foundation for grouping such cases with fetid atrophic rhinitides.

### Clinical Relationship of B. Rhinosepticus and B. Friedlander Group to Ozena.

In our previous communication on this subject we were inclined to ascribe, from the bacteriologic viewpoint, to B. rhinosepticus the sole etiologic role in clinical ozena. B. Friedlander was looked upon as a secondary invader. Although the co-isolation of B. rhinosepticus was noted in a few instances of Friedlander cases, the latter organism seemed destined to dominate the entire bacterial stage—an end reaction of immutual symbiosis. There have arisen as yet no impressions to indicate a recession from such assumption. The final solution of this problem, however, must rest upon bacteriologic experimentation. These have not as yet been sufficiently advanced to warrant definite conclusions.

Certain clinical results, also, tend to emphasize the etiologic importance of B. rhinosepticus in that improvement usually occurs in cases of fetid ozena from which both organisms are isolated, under rhinosepticus vaccine treatment, but no results have ever been obtained with even an autogenous Friedlander vaccine. Even frank Friedlander infection cases at times respond gratifyingly to rhinosepticus vaccine although the marked failures and most discouraging results have always been in cases of seemingly pure B. Friedlander infection with extensive accessory sinus disease. Again, in a considerable number of instances, initially giving plate colonies of B. Friedlander and absence of B. rhinosepticus, was the rhinosepticus organism isolated after several injections of this vaccine. We offer in explanation of this occurrence the theory of focal activation of a deeper or higher seat of infection.

Reference has been made to the common existence of Friedlander bacilli in rhinitis when associated with accessory sinus involvement. This organism often appears to persist in pure culture under such conditions. We are, though, mindful of the probable priori sinus invasion by rhinosepticus, or that secretions are mechanically arrested through atrophy and enlargement of the nasal chambers, creating an ultimate Friedlander habitat.

Persistent negative B. rhinosepticus findings in non-fetid atrophic rhinitis, chronic hypertrophic rhinitis, sinusitis without odor and normal individuals, is one of the strongest arguments presenting itself in determining the etiologic relationship of this organism. In the past year a consistent endeavor has been made in determining, from a clinical standpoint, whether or not this organism might be but a simple saprophytic entity. Bacteriologic findings have been uniformly negative. Of interest also is the paucity of bacteria noted in non-fetid nasal affections and in the nasal cavities of normal individuals.

We have as yet pursued no definite lines of investigation in determining the modus and means of infection. We are, though, inclined to support the original contention of Perez in that the disease is acquired from dogs. This hypothysis is apparently negatived in the clinical charts herewith presented, nevertheless we feel that the question of animal pets has been too closely answered in that through the general domestication of the dog a much more liberal contact has been indulged in. An interesting case in point of contact and possible bacterial communication has come to our attention. A mother and child presented themselves with definite clinical ozena. On interrogation it was found that a pet dog was afflicted with a chronic nasal catarrh, which condition preceded that of the humans. Bacteriologic examinations revealed a B. rhinosepticus infection in the mother and child and a B. bronchosepticus infection in the dog —apparently a distemper carrier. We are as yet compelled to reserve comment upon these findings.

We do not find the isolation of B. rhinosepticus encumbered with any difficulties and are becoming more and more rigid in our demand for the demonstration of this organism in sustaining the diagnosis of true ozena. During the year, 343 specimens were submitted for bacteriologic study. These cases embrace the entire gamut of intrarhinal pathology as well as a certain group of individuals with normal nasal mucous membranes. Of this number the cultures from 126 cases showed B. rhinosepticus—all clinically true ozenas—and 76 cases, a portion of which were apparently true ozena, gave exclusive B. Friedlander cultures. Of twenty-two of the B. rhinosepticus culture cases were cultures mixed with B. Friedlander. B. pyocyaneus was present in four instances. The presence of this organism makes the isolation of other organisms difficult and often impossible. One hundred thirty-seven cases were negative to either B. rhinosepticus or B. Friedlander and were often sterile.

TABLE I.

BACTERIOLOGIC SUMMARY OF NASAL SECRETIONS EXAMINED DURING THE PAST YEAR, INCLUDING CASES OF TRUE ATROPHIC RHINITIS, NON-ATROPHIC RHINITIS AND NORMAL INDIVIDUALS.

| | | |
|---|---|---|
| B. Rhinosepticus: | | |
| Alone | 104 | |
| With Friedlander | 22 | |
| Total | — | 126 |
| B. Friedlander: | | |
| Alone | 76 | |
| With Rhinosepticus | 22 | |
| Total | — | 98 |
| B. Pyocyaneus | | 4 |
| Negative to B. Rhinosepticus, or B. Friedlander or Sterile Cases | | 137 |
| Total | | 343 |

## Vaccine Therapy.

We deem it a far cry to attempt to establish the etiologic relationship of a microorganism to a certain disease through clinical results obtained by injections or suspensions of the killed organism in question. The fundamental purport of our investigation has been one of etiology. On the other hand, whilst appreciating the caution necessary in attempting to establish the causal relationship of a certain microorganism to a definite morbid process, we have inclined to vaccine therapy in the hope of finding some more potent weapon of combat against this obnoxious disease, to strengthen our heretofore limited and unpromising measures of relief.

Whether or not the rhinosepticus vaccine is specific, or through the parenteral digestion of a foreign protein the colloidal dispersion renders a mobilization of ferments, thereby elevating the titre of infection resistance, is a moot point in biology still to be determined. We are dealing with a focal infection, distinctly insidious and chronic. Intravenous vaccine injections have produced a transient leucocytosis and constitutional reaction without in any way influencing the intranasal conditions. Subcutaneous injections have, on the contrary, afforded us promise of amelioration of the manifestations of a heretofore hopeless malady. Clinical improvement is the common response. Clinical cures have been rare in that relapse after discontinuance of treatment is not infrequent. Bacteriologic cures, except in one or two instances, have never been ob-

tained. Ample drainage in conjunction with vaccine administration appears to be the most fruitful method of treatment, where there is accessory sinus involvement.

Incident to a scattered and dissociated therapeutic investigation, and with an aim at uniformity, a certain empiricism must of necessity obtain. In supplying a vaccine quite generally throughout the country for the treatment of ozena, we have prescribed such outlines of administration, in reference to dosage and injection interim, as were suggested by our own experience. We have advised as an initial injection about 200,000,000 killed organisms, increasing this and subsequent doses by 200,000,000 every fourth day. Such dose first preceding that inductive of constitutional reaction is termed the dose of tolerance. This usually occurs between the administration of the 600,000,000 to 1,000,000,000 dose. The interim of injection is now lengthened to seven days and the doses increased about 250,000,000.

In the main, the treatment of our own cases has followed this outline. We have found it necessary, though, to exercise a considerable watchfulness of the patients' individual variation in regards to idiosyncracy, susceptibility and toleration. Some patients seem unable to tolerate small doses of vaccine but after a persistent endeavor take relatively large doses without discomfort. Some show no improvement through this method of therapy until excessively large doses are reached, whilst we have noted an occasional patient respond clinically only to small doses and with marked and discomforting nasal manifestations on large doses. In other words, it is our impression that the vaccine treatment of ozena parallels that of most other insidious and relatively benign infections. We have not as yet a suitable measure or index of immologic response other than our clinical sense. The vaccine therapy of ozena is encumbered, and the negative results encountered, like unto the chronic infections in general.

### Abstract of Case Reports.

The responses to a questionaire submitted by us to those having used the vaccine prepared by us during the past year were on the whole very satisfactory. About 35 records were filled out with sufficient detail upon which reliable conclusions could be based.

It would appear from these answers that ozena is a widespread disease, prevalent in all parts of the United States, afflicting rich and poor, children and the middle aged, those in urban and rural

communities, and showing no choice or disregard to occupation and vocation. Nor does it appear that the disease has a predilection for those living in a questionable hygienic environment.

From various sources we gather the impression that many cases of ozena have been neglected owing to unpromising measures for relief at disposal, and that the ozena patient was rather looked upon as an unwelcome visitor. Latterly there appears to be a distinct revival of interest in this subject as well as a keener appreciation of the importance of associant accessory sinus involvement. Liberally discounting our own zeal in advancing a vaccine for the treatment of these cases and the general enthusiasm engendered by any new therapeutic measure, it nevertheless appears that the results of treatment by means of vaccine have been on the whole satisfactory and in some instances brilliant.

A general survey of the clinical comments of other observers and their vaccine results check remarkably well with our own experiences. Clinical cures have been claimed with reservation and many relapses have been noted. In table attached are shown the end results of vaccine treatment of cases reported by us in our previous communication.

Briefly summarizing the salient features of clinical ozena we may conclude as follows:—

*Family history:* There is an apparent negation to the fact of the frequent infection of other members of the same family.

*Infection from animals:* As charted it appears that the possibility of infection from lower animals, especially dogs, is decidedly remote. We feel, however, that too strict an interpretation of animal contact and animal pets has been indulged in that the domestication of the dog is a most common custom in this country. We feel that, upon a bacteriologic basis at any rate, the original contention of Perez will ultimately be proven to be the truth.

*Odor detected by patients:* The previously held belief that patients with ozena of long standing ultimately loose the ability of detecting their own odor is apparently incorrect.

*Characteristic odor:* The odor from ozena patients is so variously described that it appears impossible to correctly qualify it. We have at times been inclined to note an odor as "not characteristic" and be supported by negative bacteriologic findings. On the other hand, the organism of Perez has been isolated when no odor whatsoever existed. The inclination is to ascribe the odor, both in character and degree, to the extent of atrophy and possibilities of drainage rather than to the products of the activities of the causal micro-organisms.

*Characteristic crusts:* There are no characteristic crusts in ozena. The type depends upon the age, extent of the process, and the mechanics of the intranasal condition. In the very old, highly atrophic cases, the flaky white crusts simply represent the normal condition for a given amount of atrophy.

*Atrophy:* In old cases atrophy is almost invariably bilateral. We have, however, every reason to believe that a unilateral ozena can exist.

*Accessory sinuses:* In 85% of our cases was involvement of the accessory sinus more than probable. This point has been discussed elsewhere in this paper. We think it highly probable that in every case the sinuses are invaded by the offending organism and no form of treatment can be considered which does not give proper weight to this fact. The frontals, because of the difficulty of examination, we know nothing about, the other sinuses seem about equally involved.

*Clinical probabilities before bacteriological examination:* It would seem that there is a certain clinical complex characteristic of bacillus rhinosepticus ozena. In our 26 cases, judged before the bacteriological study, only three mistakes were made as to the true bacteriological nature of the complaint. This would seem to indicate that the ozena due to bacillus rhinosepticus and Friedlander group are clinical entities and should be classed as such in contradistinction to marked atrophic rhinitis with odor and crusts due to syphilis, tuberculosis and other constitutional diseases.

*Vaccine Injection*—The average number of injections used ranges around 15. The improvement was usually noted by the fourth injection and continued up to the 12th or 15th. From then on it seems to settle itself down into a long process of supplying the patient with enough antibodies to keep the disease well under control and in some cases can be continued indefinitely with good results.

*Character of Reaction*—With proper vaccines, given in proper doses the reaction is never severe. Marked symptoms are to be avoided. The best results are obtained where one remains just under the reaction point. The seven day interval, after the proper dose has been reached, seems to be the most convenient and most useful.

*Results*—Although our main interest in the study of ozena during the last three years has been largely confined to the question of etiology and clinical pathology, general interest seems to be in the results. The beneficial results of vaccine treatment were marked in the great majority of cases. End results or the permanency of clinical cure have been difficult to establish owing to the fact that certain cases were lost from observation when crusting and odor disappeared. Thus in 23 cases treated in Chart II of our last year's report, 13 noted as clinical cures did not return for treatment.

*Summary of 1916 Results, Still Under Observation*—Case 1, the priest, who was obliged to douche his nose every few hours in order to carry on his confessional work, has had no return of symptoms as far as crusting and headaches—but from time to time notices a slight odor, easily controlled by an occasional douche.

Case 2, remains a clinical cure, without crusts and odor and never has to douche the nose. The B. rhinosepticus can always be demonstrated.

Case 3, one of the most evil smelling of this series, remains clinically cured without any local treatment. This person could not be admitted to the reception room on account of the foulness of odor.

Cases 10, 11 and 12, mother and two children, remain about the same. The treatment has been almost continuous throughout the year. Because of positive complement fixation test for tuberculosis it is possible we are dealing with a latent tuberculosis which may account for the poor result.

TABLE II.

SUMMARY OF CASES REPORTED BY US IN 1916 WITH COMMENT ON CONDITION A YEAR LATER.

| No. | Old No. | Name | Age | Sex | Nationality | Referred by | Clinical Diagnosis | Bacteriologic Findings | Agglutination | Number Inj. | Crusts | Odor |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 1 | Adolphus | 28 | M | German | Dr. Victors | Ozena | Perez | + | 20 | | |
| 2 | 2 | Alpert | 22 | M | Russian Jew | Dr. Richards | " | " | + | 10 | | |
| 3 | 10 | Miranda, Mrs. | 37 | F | Spanish | Poly | " | " | + | 9 | | |
| 4 | 11 | Miranda, J. | 11 | M | Spanish | Poly | " | " | | 9 | | |
| 5 | 12 | Miranda, H. | 12 | M | Spanish | Poly | " | " | | 9 | | |
| 6 | 14 | Plant | 20 | F | American | Office | " | " | + | 19 | | |
| 7 | 20 | Sponner | 35 | M | American | Office | Atr. Rhin. | " | — | 5 | 0 | 0 |
| 8 | 21 | Presecella | 22 | F | German | Office | Pan Sinusitis | " | — | 19 | + | + |
| 9 | 22 | Weber | 27 | M | American | Office | Ozena | " | + | 14 | + | + |
| 10 | 8 | Kelleher | 17 | F | German | Dr. Victors | " | " | + | 20 | + | + |

| No. | Results in 1916 | Results in 1917 |
|---|---|---|
| 1 | No crusts, no odor. Clinical cure. Under treatment. | No vaccine treatment for months. No crusts. Slight odor at times, easily controlled by salt water douche. Sinuses not investigated. |
| 2 | No crusts, no odor. Clinical cure. | Clinical cure. |
| 3 | No crusts, no odor. Under treatment. | Results very unsatisfactory, constant treatment during year. Odor decidedly improved but still marked at times. No local treatment. |
| 4 | No crusts, no odor. Under treatment. | Results very unsatisfactory, constant treatment during year. Odor decidedly improved but still marked at times. No local treatment. |
| 5 | No crusts, no odor. Under treatment. | Results very unsatisfactory, constant treatment during year. Odor decidedly improved but still marked at times. No local treatment. |
| 6 | No crusts, no odor. Clinical cure. | Remains clinical cure. See detailed report in body paper. |
| 7 | Ozena laryngis? Great improvement. | Cured. |
| 8 | No crusts, no odor. Clinical cure. | Remains clinical cure. See detailed report in body paper. |
| 9 | No crusts, no odor. Clinical cure. | Remains clinical cure. See detailed report in body paper. |
| 10 | No crusts, no odor. Clinical cure. Under treatment. | Persisting slight odor. No crusting as long as occasional vaccine. |

Case 14, a young woman whose engagement was broken on account of the odor, has remained a remarkable clinical but not a bacteriological cure. The girl married her former affianced and considers herself cured.

Case 21, Sister P., three years under treatment for pansinusitis, remains clinically but not bacteriologically cured. She has had no vaccine treatment for the past year.

Case 22, dentist, was obliged to limit his practice on account of the odor, remains a permanent clinical cure. He has had one or two short vaccine treatments throughout the year and still gives a positive B. rhinosepticus.

Thus it will be seen that every case in Chart II reported as clinical cures have remained so, a very satisfactory record. The constant presence of the B. rhinosepticus shows that these cures are not permanent but that antibodies must be supplied indefinitely in the form of small doses of vaccines, repeated at intervals, for an indeterminate period.

Chart III contained 9 cases, 8 Friedlander ozena treated with our vaccine, 7 of the 9 noted as clinical cures, did not return for further treatment, the other two have remained entirely well without vaccine or local treatment during the year. The Friedlander is still present in cultures.

*Analysis of Results of New Cases Treated Since Last Report*—26 cases are reported in our new series (Table III). Six were untreated. Five did not improve. Six cases improved as the results of operative drainage on the sinuses and no credit was given to the vaccine. In the nine remaining cases great improvement or clinical cures are noted.

Of the five cases showing no improvement, only one was a true B. rhinosepticus ozena. This case received but three injections and did not return.

The remaining nine cases form a happy paragraph in the therapy of this disease. The improvement was marked, in some cases very striking, the crusts and odor rapidly disappeared. The frontal headaches invariably cleared up after the third or fourth injection. Weight and general condition improved. These and the last year's series have been carefully and fairly studied, we see no reason to change our conclusion of last year (No. 5), that "At present mixed vaccines made from various strains of the Perez bacillus (B. rhinosepticus) is the most practical method of treatment which is now available."

## DISCUSSION.

**Dr. William R. Murray,** Minneapolis, Minn., said: "Before discussing the etiology of ozena, I wish to congratulate the essayist on the great amount of work he has done in connection with this subject. The etiology of ozena has been an unsolved problem and the therapy of this disease has been equally unsatisfactory. It is only by an intensive study of ozena that we are going to accomplish anything. I am not as optimistic, however, as Dr. Horn is in regard either to our knowledge of the etiology of ozena or its treatment.

"Two years ago Dr. Larson and I published the results of our study in regard to the Perez bacillus being the cause of ozena, and further study, bacteriologically and clinically, has not enabled us to change our opinion. You are all familiar with the literature on the bacteriology of ozena, but I would like to call attention to a few points in connection with the published work of Perez and Hofer. In our work we were unable to isolate the Perez organism from ozena cases, and an analysis of the work published would indicate that there is considerable doubt that this bacillus has ever been obtained directly from the patient.

Hofer states that it is very difficult to isolate from the human, and, while he rather leads us to infer that he has isolated it, he does not positively state so. The Perez bacillus is described as Gram-negative bacillus. This description, however, does not identify the organism. It is only a morphological description and there are a large number of Gram-negative bacilli which can be isolated from ozena cases, and these different bacilli cannot be differentiated morphologically. It is only by cultural and serological tests that they can be identified. In our work, two years ago, we were unable to isolate a Perez organism from our ozena cases, and further work, since that time, has not changed our findings.

"Where does Hofer obtain his organism, which he claims is the cause of ozena? He goes to the rabbit's nostrils for it. He previously injects, into the ear vein of the rabbit, a mixed culture obtained from an ozena case. He then obtains, from the nostrils of the injected rabbit, the Perez organism. Was the Perez bacillus present in the nostril of the ozena case, or did it previously exist in the rabbit's nostril? It seems strange to us that if this organism is the cause of ozena and is present in ozena cases it can not be isolated from the patient. There is present at times in the nostrils of some animals, including the rabbit, an organism, the B. bronchisepticus, that has all the morphological and cultural characteristics of the B. Perez. These bacilli, the B. bronchisepticus and the B. Perez, are probably identical. Dr. Horn, in one of his recent publications, states that the B. bronchisepticus and the B. Perez give positive complement fixation tests to the same serum. This would prove their identity. The B. bronchisepticus is also present, at times, in dogs and is greatly increased in the presence of a nasal infection in dogs, and in cases of 'snuffles' in rabbits it is frequently present.

"There are a number of organisms present in the nostrils of ozena cases that resemble, morphologically, the Perez bacillus. One of the most frequently present Gram-negative bacilli is the proteus, as shown in our report in 1915. This organism appears, microscopically, identical with the Perez, except that it is motile. Culturally it is quite different. Dr. Horn describes his organism, which he isolates from ozena cases, as being motile, and assumes that it is the Perez organism. The Perez bacillus is non-motile.

"Serological tests also disprove the theory that the B. Perez is the cause of ozena. It is generally agreed that ozena cases never give any serological reaction, such as the agglutination or complement fixation tests, with the Perez organism. All investigators agree that the animals treated with the Perez bacillus respond promptly in the production of both agglutinin and complement fixation antibodies. In view of the fact that antibodies are so readily formed against the microorganisms in treated animals, why is it that blood of ozena patients never contains such antibodies until after having been vaccinated?

"Regarding the results of vaccine treatment upon the course of ozena, there seems to be no unanimity of opinion at the present time. While some investigators report favorable results following the use of Perez vaccines, others using the same vaccine find the treatment does not influence the course of the disease. Personally I have used Perez vaccines prepared from cultures received from Hofer, also vaccines received from Dr. Horn and Dr. Victors, as well as ozena vaccines supplied by commercial houses, and the results have not been encouraging enough to warrant any belief that the vaccine treatment is effective. Even though a few cases do show some improvement under vaccine treatment, this must not be regarded as conclusive evidence that the microorganism from which the vaccine is prepared is the specific cause of ozena. We not infrequently see what appears to be improvement in certain conditions where a non-specific vaccine is used. For instance, Hodgkin's disease not infrequently improves temporarily under vac-

cines, and it seems to make little difference from what germ the vaccine is prepared. Favorable results have been reported from the use of Lowenberg-Able vaccine in ozena.

"The fetid odor of ozena is probably caused by proteolytic organisms, growing upon the crusts, secretions, and destroyed tissues, and these organisms probably bear no relation to the etiology of ozena. At the present time, it would seem that there is insufficient evidence to justify the conclusion that ozena is caused by any specific organism."

**Dr. Wolf Freudenthal,** New York City, speaking from the clinical point of view, had come to the conclusion that the primary cause of ozena was atrophy of the mucous membrane, infection being the secondary cause. Whether the organism involved were the bacillus of Friedlander or of Perez made no difference. The fact that Dr. Horn found the bacillus of Perez in the sinuses brought up the point of focal infection, to which Gruenwald, Hajek, and others, had called attention many years ago. In his work with tuberculous patients he had found all sorts of organisms in their sinuses. The investigation of vaccine-therapy in connection with ozena should be continued.

**Dr. John Horn,** New York City, had never had such good results in these discouraging cases as when he had used Dr. Henry Horn's vaccine.

**Dr. Joseph H. Abraham,** New York City, reported six cases, five private and one from the clinic. He could not get a bronchisepticus vaccine in any case, and the Friedlander in only two. The vaccines gave only fair results. He used a stock vaccine in one case. In one case complicated by empyema of the antrum, he proposed to operate in a few days.

**Dr. Thomas J. Harris,** New York City, had been carrying out for a considerable time, at the Manhattan Eye and Ear Infirmary, a careful study of this subject. He had taken the vaccine side and Dr. Coffin the operative side. His work had been absolutely negative. When he had failed, Dr. Coffin had taken the cases, and in two-thirds of them had obtained a cure by opening and draining the sinuses.

**Dr. Horn,** in closing the discussion, was gratified to know that skilled bacteriologists, such as Dr. Dwyer and Dr. Murray, had investigated his work. He reiterated the fact that he had approached the subject as a clinician, not as a trained bacteriologist. The latter part of the subject had been under the control of Dr. E. Victors, of San Francisco, a trained bacteriologist.

Of the series of cases reported last year, ten remained under treatment. In one case, that of a Catholic priest who had found it absolutely impossible to continue his work in the confessional, no treatment, so far as the vaccines were concerned, had been given during the past year, and the patient had been able to continue his work. This was but an example of the marked clinical improvement in these cases. Full details would be found in the Transactions.

# ABSCESS OF THE LUNG FOLLOWING OPERATION ON THE TONSILS AND UPPER AIR TRACT.

By CHARLES W. RICHARDSON, M. D., Washington, D. C.

It seems unnecessary for me, in this paper, more than to call attention to the members of this Society to the papers that I have written bearing upon this subject before other organizations as I feel that they have been quite generally read. On March 20, 1912, I believe I first called attention to this condition as a complication of tonsillectomy. Since the publication of my paper on "Abscess of the Lung, Following Operations on the Upper Air Tract," in the Laryngoscope, July, 1916, I have received a number of communications from observers throughout the country, stating that they had met with this untoward experience in their tonsillectomy work. It having been established that this serious complication is not now an unusual sequella of operative work, it therefore, becomes incumbent upon us to ascertain the manner of its occurrence, and to consider wherein we may adopt alterations in our methods, or technique, by which it may be prevented.

To all observers, it must be patent that this complication has largely come under our observation since we have adopted the more radical operation of tonsillectomy in dealing with enucleation of tonsils. It must also be patent to every one that while we do a great many more radical operations upon the sinus than formerly, in which there is free pus, granulation tissue, and infective debris, which must in certain amounts gain ingress into the air tract, yet we do not find that lung abscess is an increasing factor in these operations.

What is the probable etiological factor that enters into the causation of this complication?

Under the various causes assigned as the factors in producing pulmonary abscess are:

First, General anesthesia.

This is only a remote factor in that the anesthetic irritates the pulmonary mucous membrane and renders it more susceptible to the invasion of pathogenic germs that may have gained ingress, and, through rendering the patient unconscious, it lowers the expulsive reflex, thus lessening the great natural defense to the protection of the air tract.

Second. The want of thorough examination and preparation of the patient.

This factor should never enter into the causation. I was astonished in my reading of the report of the New York Academy of Medicine, Section on Laryngology and Rhinology, meeting of January 26, 1916, of the discussion on this point. There is and can be no excuse for the want of examination and preparation of patients about to enter into operative procedure upon the upper air tract. My patients are always examined physically. The orders for the preparation are always written out and given to each patient with injunction to follow explicitly. I have never had a patient to vomit on the table. The bowels are always freely opened, and they have no food for at least 18 hours before the operation. The urine is always examined.

Therefore after a most thorough consideration of this subject, it seems to me that the method of invasion is either indirect through the lymphatic or venous system from the wound surface, or directly through the inspiration of pus or bacterially laden caseous material into the pulmonary tract. By whatever method we perform this operation there is more or less squeezing of the tonsils so that contained pus or masses of cheesy debris is forced out from the substance of the tonsils or from the diseased crypts. Even while making the incision to liberate the folds of the tonsils, we have on numerous occasions observed pus, more particularly cheesy concretions, floated out on the surface of the tonsils. This occurs almost as frequently with children as with adults. By whatever method we perform the operation we necessarily open up numerous lymphatic vessels and moderate size veins and leave a deep wound surface, which is bathed in the abundant flora of the faucial cavity.

If these are the two important factors, which I believe they are, we should make all effort to minimize their effect by lessening the amount of trauma as far as our operative skill will permit and through adopting all methods to prevent the ingress of pus or cheesy material from the tonsils into the pulmonary tract.

I had hoped through the various improvements of my own technique to exclude this complication from my operative risks but my work during the past year has shown me that I have not succeeded. In spite of my depression of the patient's head, through the raising of the foot of the operating table 35′, and the use of the suction ether apparatus of the Beck-Mueller design, which has always worked most satisfactorily, I have had one abscess

formation. One interesting feature to me in this connection is the fact that I have never seen an abscess of the lung following an operation upon the upper air tract in a child under fourteen years of age. In the Children's Hospital, in Washington, in which the entrance age is under twelve and wherein I do for six months all the open ward, and throughout the year many of my private children patients' operations for tonsillectomy, and where we do not have the advantage of the suction-ether apparatus, I have never had a pulmonary abscess.

All my adult operations are done at the Episcopal Eye, Ear and Throat Hospital, as well as some of the older children under the greater advantages of position and suction. The number of my operations done in each institution is about equal. Is there some anatomical relation in the fauces, or is the resistance power in the lungs in a child greater, which renders it less susceptible to this type of infection than the adult?

From the fact that children seem totally unsusceptible to this type of infection, from the fact that infection occurs as a result of submucous resection, which is an absolutely clean operation; and that it does not transpire with any degree of frequence after opening up such pus bearing fields, as the nasal sinuses, I am still inclined to believe that this complication occurs in the fair proportion of cases by infection through the lymphatic or venous channels. Another strong factor that points to this manner of infection is the fact that most of the abscesses that I have seen radiographed have apparently developed between the lobes of the lung, probably from the inter lobular lymph spaces.

With these facts before us should we not seek to modify our operative procedure upon the adult? Is the patient under general anesthesia more susceptible to this form of infection than under local anesthesia? In the occurence of faucial and deep cervical infection after local anesthesia as frequent and as dangerous to the patient as the occurrence of occasional lung abscess in the general anesthetized adult? The above question can only be answered after a thorough survey of the situation and upon better knowledge of the advantage offered through the two methods than we now possess.

*Symptoms of Lung Abscess following Operation upon the Upper Air Tract—*

The earliest and most characteristic symptom is that the patient does not make a favorable convalescence after the operation. The case progresses favorably with regard to local manifestations, but

the patient seems languid, somewhat dispirited, and inclined to remain abed. Usually from the fourth to the tenth day positive evidence of pulmonary invasion makes itself manifest. We have irregular septic temperature, sharp pain in the lung and violent paroxysmal coughing. Occurring at the same time or shortly thereafter we have the pathognomic evidence of pulmonary abscess in the expectoration of a free pus which has a foul odor. This foul odor is intensely distressing to the patient and is noticed by them, not only with the expectoration of the pus, but also during coughing. In some cases the odor is noted by the patient some hours before the expectoration of pus. In the early stages, and frequently throughout the case, the foul odor is only noted by the patient. The physical sign at this stage is very meager, depending upon the size and location of the area involved. These signs are dullness over the area affected, diminished breath sounds and crepitant or sub-crepitant rales. There is no doubt that some cases at this stage are supposed to be ether pneumonia. With the development of the abscess, we have characteristic symptoms manifest, viz., the expectoration of pus, pain, showing of blood to actual hemoptysis, and foul odor. In the several cases which I have had under my observation, although the patient complained bitterly of the odor, the odor of the breath was never noticed as disagreeable, although the expectoration from the same patient had a distinct, unpleasant odor. The cough persists, nausea is frequently present, the fever becomes septic, the fingers club and the patient gradually loses flesh. The physical signs at this stage are those of lung abscess. Radiograph taken at any time will show the graphic evidences of the abscess.

Treatment.—The management of pulmonary abscess may be considered under three classifications, viz., medical, or expectant; surgical; or compression of the lung by artificial pneumothorax.

In a small percentage of cases when the abscess is located in the upper lobe of the lung and the drainage is into a large bronchus, and where the case is apparently following a favorable course the medical expectant treatment may be followed. It is well to have in mind the fact that the percentage of mortality is very great by this line of treatment, approximately 60%, and therefore it must not be pursued for too long a period.

The surgical method, consisting in rib resection, fixation of lung and drainage of pus gives a much better result than medical treatment, with a mortality of approximately 30%. The more radical

surgical procedure of excision of portions of the lung containing abscess cavity, indeed of one or more lobes, has been attended with exeellent results. The surgical treatment has offered the best results heretofore, although with a fairly high mortality and frequently a prolonged drainage before the case is brought to resolution.

*Artificial pneumothorax*—By this method we have apparently the ideal, rational and most acceptable method of treating acute pulmonary abscess. As is well known the method has been for a long time used with more or less marked success in the treatment of chronic pulmonary abscess. Dr. W. D. Tewksbury, superintendent of the Washington Tuberculosis Hospital, I believe was the first to suggest its use in acute pulmonary abscess, although he gives credit to Dr. A. E. Greer, to priority in the publication of a single case of interlobar empyema following pneumonia treated by compression of the lung with nitrogen. Dr. Tewksbury in his production of artificial pneumothorax employs air instead of nitrogen. Dr. Tewksbury in a paper, read before the Medical Society of the District of Columbia, entitled, "Acute Pulmonary Abscess treated with Artificial Pneumothorax," November 8, 1910, gives the results of treatment in his first two cases. He has kindly furnished me with the data and results obtained by him in treating acute pulmonary abscess since the publication of his above mentioned paper. In the past ten months he has seen ten cases of acute pulmonary abscess, nine of which followed operative treatment upon the upper air tract, by eight different operators, in eight of which he employed artificial pneumothorax. In two cases expectant treatment was attended with resolution. It is interesting to note that these cases were operated upon by eight different operators, each employing a slightly different technique, so that the result can not be assigned to a universally employed technique. These results also demonstrate quite a large proportion of lung abscess for a community as small as Washington, and bears out the statement that I made in my paper read before the American Climatological and Clinical Society, May 9, 1916, "but while these remote lesions are rare, I am convinced that they are not so infrequent as one would judge from the domain of laryngo-rhinology."

Dr. Tewksbury's conclusion is as follows:

Of the eight cases treated by artificial pneumothorax, six were acute lung abscesses of a month or less duration. Of this number four recovered, one is recovering (at this writing well) and one

died. Two were chronic lung abscesses of over a year's duration. Both improved but were not cured.

It is apparent that in order to get cures in these cases, it is necessary to use pneumothorax early while the abscess is soft and compressible.

# RESULTS OBTAINED BY TONSILLECTOMY IN THE TREATMENT OF SYSTEMIC DISEASES.

By DANIEL W. LAYMAN, M. D., Indianapolis, Ind.

The most important work in medicine in the last decade is the elucidation of the relationship of focal infection, especially that of diseased tonsils, to systemic disease. That the tonsils play an important part in various infections was demonstrated years ago by laryngologists. In this connection I refer you to such men as Wood, Goodale, Wright and others, whose research as far back as the nineties shows that the laryngologist must be recognized as the pioneer in this work. However, this early enlightenment of the individual worker in tonsilar infections did not at first engage the attention of general medical practitioners. But when, within the past few years, the real significance of tonsilar infection was demonstrated, through the bacteriological studies and experiments of laboratory workers, cooperating with clinicians, the medical world recognized a new and a very important factor in medicine. Rosenow's newer bacteriology, and the excellent work of men like Billings, Davis and Jackson, with their cooperators, who worked to better advantage than could separate individuals, awakened the general practitioner to the importance of this great subject. Davis made a careful bacteriologic study of the bacteria removed from the tonsils in various diseases. He reproduced many of these conditions in animals by the injection of the bacteria. His studies included patients with endocarditis, nephritis, arthritis, etc. Rosenow proved that many ulcers of the stomach are associated with tonsilar infections. He obtained streptococci from some of these ulcers, and after injecting cultures of these organisms into animals, was able to observe gastric ulcers in them.

Today we are in the midst of a period in which tonsillectomy as a therapeutic measure in the treatment of systemic diseases is very popular. The laryngologist does not need compiled data to be told that the removal of diseased tonsils for systemic diseases is followed by improvement in the general health of the patient. Those who feel satisfied that the beneficial results are evident without enumeration of statistics are not heeding the claims, and even protests, that have appeared from time to time in both medical and lay papers; claims that the tonsil is held responsible for too many diseases, and that the beneficial effects do not justify the numerous

enucleations. These articles have not dealt kindly with the surgical procedure of tonsillectomy. Even a few responsible and distinguished medical men pronounce such criticism as most timely.

It is incumbent upon us, therefore, to inform ourselves regarding this important subject; to ascertain whether or not the pendulum has swung too far, lest the medical profession at some later period declare that we have sacrificed tonsils unnecessarily in the treatment of systemic diseases.

Sufficient time has elapsed since tonsillectomy has been so freely advocated to enable us to study the general results. Is tonsillectomy doing for these clinical conditions all that the laryngologists and the internists expected? Has the measure a substantial scientific backing?

A few months ago, in order to obtain as accurate data as possible, I submitted an inquiry to many laryngologists and a few internists throughout the country, requesting information concerning this matter. The statistics herein compiled do not make any pretension to exactness, but the information therein contained ought to serve the purpose of showing us where we are. As someone has said: "The best way to get ahead is sometimes to stop short and see where we are."

The questionaire submitted to laryngologists and internists requested information regarding the results obtained by tonsilar enucleation in cases of arthritic, cardiac, renal, and other systemic diseases. Only complete reports have been compiled. the cases reported in numbers were kept separate from those reported in percentages. Out of many responses to the query, ten reports giving the number of cases furnished available material for statistics. The ten reports include 894 cases. The list of questions in the query and the compiled data are as follows:

1. In systemic diseases where the tonsils were considered the only source of infection, in what number or in what percentage of cases did tonsillectomy produce:

| | Reported in number of cases. | Reported in percentage of cases—average. |
|---|---|---|
| (*a*) *Real cures of the systemic diseases.* | | |
| Arthritic | 262 | 68% |
| Cardiovascular | 3 | 36% |
| Renal | 21 | 81% |
| *Other conditions.* | | |
| Rheumatic group (neuralgia, lumbago, peri-neuritis, myositis, etc.) | 220 | .. |
| Chorea | 3 | .. |
| Cervical adenitis (simple) | 57 | .. |

| | | | |
|---|---|---|---|
| (*b*) | *Considerable improvement of the systemic disease.* | | |
| | Arthritic | 184 | 20% |
| | Cardiovascular | 27 | 18% |
| | Renal | 12 | 5% |
| | *Other conditions.* | | |
| | Rheumatic group | .. | .. |
| | Chorea | 17 | . |
| | Cervical adenitis (simple or tuberculous) | 51 | .. |
| | Hyperthyroidism, (goitre) | 13 | .. |
| (*c*) | *No improvement of the systemic disease.* | | |
| | Arthritic | 14 | 12% |
| | Cardiovascular | 3 | 46% |
| | Renal | 4 | 14% |
| | *Other conditions.* | | |
| | Rheumatic group | 3 | .. |
| | Goitre | 1 | .. |
| | Cervical adenitis (tuberculous?) | 12 | .. |

2. Following tonsillectomy, what percentage of cases developed:

| | | | |
|---|---|---|---|
| (*a*) | Acute exacerbation of the systemic disease: | | |
| | Arthritic | 10 | 10% |
| | Goitre | 1 | .. |
| (*b*) | A post operative low grade infection | 9 | 3% |
| (*c*) | Coexisteut infection as causative factors of the systemic diseases | .. | 28% |
| (*d*) | Nasal sinuses | 49 | .. |

Twelve reports giving the results in percentages were available for compilation. The cases reported in percentage averaged as follows: (See above.)

The answers to the questionaire, confirmed by the above figures, show that tonsillectomy in the treatment of systemic diseases, when based on rational and well founded relation of cause and effect, and when performed under the most favorable conditions, has given exceedingly satisfactory results.

The percentage as presented, especially in the arthritic class, compares favorably with statistics of other observers. Every laryngologist and those interested in the subject should be conversant with the excellent report on "The Relation of Tonsillar and Nasopharyngeal Infection to General Disorders," that appeared in the January, 1917, Bulletin of Johns-Hopkins Hospital. The Hopkins report states that "only in very exceptional circumstances should anyone subject a patient with rheumatoid arthritis to an operation for the removal of the tonsils." A composite statement deduced from many general reports and comments which I received would read that tonsil enucleation alone does not have a beneficial effect on well established conditions, viz., definite arthritic changes and deposits, as ankylosis and fixation, chronic renal disease or chronic heart and blood vessel disease, etc.

The very favorable results in the arthritic class as reported in numbers show that the operators were either most careful in the selection of cases, or else that the general health of the patients was afterwards so much better that the operators took it for granted that the arthritic conditions were also improved. The twelve per cent. reported in the "no improvement" arthritic cases is, in my opinion, a fairer statement of the results in the arthritic group. If twelve per cent. show "no improvement," what explanation can be given for the lack of improvement in this number of arthritic cases?

One observer said that real cures in these cases followed only after a postoperative use of the autogenous vaccine and local treatment. Dr. Jas. J. King, of New York City, takes a step further in the use of vaccines. He advocates taking cultures from the crypts of the tonsils before removal and making a vaccine to be given until the bacterial activity is stopped or reduced. The tonsils are then enucleated, and if necessary the vaccine is continued after operation. He recommends this method of procedure as most satisfactory, and reports a series of cures. It may be that such a course of treatment in many arthritic cases will reduce the number that show "no improvement" and lessen the chances of the development of an acute exacerbation of the arthritis disease after tonsillectomy.

Some individual comments taken from the responses of physicians who answered in a general way are mentioned here, as they add information of interest. One observer reports a large series of thyroid enlargements with pronounced tachycardia as markedly benefited by tonsillectomy. Letters from prominent surgeons who do a great deal of thyroid surgery show that there is a decided uniformity of opinion regarding the relationship between tonsilar infection and the enlargement of the thyroid and hyperthyroidism. All of them advise tonsillectomy in goitre cases that give a history of tonsilar infection, but they do not depend on the procedure as a curative measure. They differ in their views as to the time of performing tonsillectomy; some advocating it before, and others after the thyroid operation. Dr. Crile, of Cleveland, and Dr. Halsted, of Baltimore, prefer to operate on the thyroid first, especially where the symptoms of hyperthyroidism are well marked, because tonsillectomy is a trying operation for these patients. Dr. Bloodgood, of Baltimore, writes: "The tonsil infection or coexistent infection should be taken care of before attacking the thyroid, and there should be an interval of at least two or three weeks between the

tonsillectomy and the operation upon the thyroid." He also states that in 50% of the cases of toxic goitre he has found either infected tonsils or an infected nasal cavity. At the Mayo Clinic they do the thyroidectomy to relieve the symptoms, and the tonsillectomy to prevent, if possible, recurrence of hyperthyroidism in the remaining lobe.

Regarding coexistent infections, one laryngologist states: "According to my experience, some coexistent focal infection, such as nasal sinus disease or some diseased condition of the gall bladder, bronchial glands, appendix, gastro-intestinal tract, lymph nodes, teeth, etc., was present in practically all cases that showed no improvement after enucleation of demonstrable diseased tonsils." Another observer stated that diseased teeth were found in a large percentage of ward cases. He wrote: "It is our practice now to have a dental surgeon scrubbed up at the operation (the resident dentist at the hospital) who has previously examined all cases and decided which teeth should be removed."

As a result of the experiences of many surgeons now collated, we have reached certain tentative conclusions which may be helpful towards securing better results when tonsillectomy for systemic diseases is contemplated.

Tonsillectomy should not be performed closely following convalescence from an acute purulent infection, such as middle ear, nasal sinus or upper respiratory tract, or acute exacerbation of some systemic disease. Reports show that such cases have developed septicemia. In these cases it is better not only to wait for a considerable time to elapse before proceeding with tonsillectomy, but to take a preliminary culture and cleanse the tonsil crypts by suction and antiseptic applications. In some the use of vaccines may be a valuable adjunct. If these acute infections have been treated in a hospital, the patient should be discharged temporarily before undergoing the operation. Some patients need prolonged treatment with a view of establishing sufficient resistance to enable them to bear such a surgical procedure as tonsillectomy.

Hospital internes and nurses, especially in recent active service with septic cases, should be given a temporary leave of absence before undergoing tonsil enucleation. Cases of arthritic, cardiac, renal and other system conditions must not be allowed to leave the bed too soon after tonsillectomy. This precaution is not observed closely enough by many operators.

The possibility of acidosis occurring in many conditions, especially in the case of children, or of those suffering from any form of exhaustion, must be reckoned with by laryngologists. When this is suspected, a proper and continued examination of the urine for its by-products (diacetic acid or acetone) should be made before operating and if found the proper treatment for acidosis given. Some operators advocate a pre-operative treatment of acidosis in all cases. The postoperative symptoms of uremia, even when albumin is found, may mislead one in recognizing the true condition, i. e., acidosis.

Albuminuria following the removal of the tonsils has been attributed to ether narcosis, the ether supposedly acting as an irritant to the kidneys. This theory cannot be held tenable in view of the bacteriological and chemical findings in the examination of the urine in many cases of tonsillectomy. A recent report by Dr. Ruddel, anesthetist at St. Vincent's Hospital, Indianapolis, cooperating with Dr. Scott Edwards, shows that practically as many cases of albuminuria followed tonsillectomy where a local anesthetic was used. They attributed the condition to the fact that a certain amount, perhaps a considerable amount of infective material, is squeezed out of the tonsils during the course of the operation and is injected into the circulation. That ether seemed to be a negligible contributory cause of albumin was shown in the study of several hundred other surgical conditions. They also observed that, where albumin was found prior to the operation, it cleared up in a great number of cases after a few days rest in bed before tonsil enucleation, and that the kidney disturbance after the operation was less severe. This I also observed in a few of my cases. Rest in bed for from one week to ten days, and a restricted diet should be the proper procedure in all cases of albuminuria found prior to the operation.

Conservatism in tonsil enucleation in early childhood has been the practice of most laryngologists. The position of many laryngologists in this respect has been adopted and maintained, no doubt because of the general belief that the tonsil should be respected as a functionating organ, especially in early childhood development. The reports of competent pediatricians, and of medical men connected with contagious disease hospitals, after years of clinical experience, show that the tonsil is a greater menace to health in early childhood than is generally recognized. In answer to a letter of inquiry regarding this phase of the tonsil problem, I received a number of

replies from competent pediatricians. The following letter from Dr. La Fetra, of New York City, is typical of the attitude of all concerning the tonsilar question:

"In answer to the inquiry in your letter of March 27, I write to say that I have never been able to notice any abnormality in growth or development in those boys and girls whose tonsils were removed in early childhood. Whatever advantage the tonsils may have physiologically, it would seem that nature has provided sufficient other tissue of the same sort to take up vicariously the work of their tonsils when these are removed. As regards their susceptibility to disease, I would say that children whose tonsils have been removed are much less susceptible to respiratory and gastro-intestinal disturbances of all sorts. Moreover, when they have measles, scarlet fever or whooping cough, the disease is much less severe than it would have been had their diseased tonsils been present."

Dr. Henry Hall Forbes' paper "The Removal of the Tonsils as a Prophylactic Measure" presented last year before the Medical Society of the State of New York, was timely. It showed the attitude of the pediatrician, health officer and other medical men regarding phases of the tonsil question concerning which there has been a divergence of opinion among members of the medical profession. The present investigation confirms the information Dr. Forbes has given us in that excellent paper. Dr. Forbes shows from the reports of health officers that from eighty to one hundred per cent. of the contagious disease cases have tonsils: also that tonsils were nearly always present in the cases which developed complications. Dr. Henry L. Lynah, New York, writes: "In our Contagious Disease Hospitals the percentage of admissions in whom tonsillectomy has been performed is from two to four per cent. The other ninety-six to ninety-eight per cent have tonsils in various degrees of size."

Regarding the association of the tonsils and dentition, one occasionally reads in medical literature that one of the functions of the tonsils is an association with dentition, as the tonsils become swollen and inflamed with the eruption of the teeth, and when the tonsils are removed in infancy and childhood there follows a deleterious effect on dentition. This question was submitted to twelve of the best orthodontists and oral surgeons in America, and they denied, without exception, that early removal of the tonsils had any effect whatever on the development of dentition. The answer to this question from Dr. Frederick B. Noyes, of Chicago, is as follows:

"I have never seen any indication that the early removal of tonsils had any effect whatever on the development of dentition, and I know of no evidence that tonsils under normal conditions are in any way connected with the development."

In view of these reports and the attitude of the most prominent pediatricians and health officers in the country, should we not seriously consider the removal of the tonsils as a wise prophylactic measure in early childhood in many more cases than formerly? Surely this will be an advance in preventive medicine and will thereby lessen the number of tonsillectomies that have to be performed in adolescence and adult life in the treatment of well established systemic diseases.

The author wishes to express his appreciation for the valuable responses from many members of this Society and others which made this paper possible.

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## DISCUSSION.

**Dr. Lee Wallace Dean,** Iowa City, Iowa, said that in his service he had had one case of pulmonary abscess following an operation on the throat of a child five years of age. The diagnosis was confirmed by the postmorten examination.

He did not agree with Dr. Richardson as to the advisability of having patients fast for six hours previous to tonsillectomy.

There had just been completed in the Chemical Research Laboratory of the Department of Medicine at the University of Iowa, an investigation of acidosis in connection with infection. The object of the research was to determine whether children with a chronic infection of the tonsils were more susceptible to acidosis than those whose tonsils were not affected. It had been found that starvation of children with chronically diseased tonsils would always produce acidosis. In short, in one instance the reaction was alarming. In view of these findings he scheduled his operations for early in the morning, and fed the children abundantly on carbohydrates late at night. As soon as possible following the operation, carbohydrates were given.

**Dr. Sylvan Rosenheim**, Baltimore, Md., said many difficulties had been encountered when the practice was started of taking out tonsils by dissection instead of tonsillectomy. The most important of these difficulties was hemorrhage, and when that was eliminated the problem seemed to be solved. Since reading the articles by Richardson, Coakley, Manges, and others, on the subject of abscess, another view of the question was necessary. He had had no experience with abscess of the lung or sepsis, perhaps because of the technic employed. He operated with the head low, in order to keep the field perfectly clear of the caseous matter. The failure to do this doubtless accounted for many lung infections.

In considering results one must consider the types of cases operated on,—whether they were mechanical cases, with obstruction to breathing and with effects on the hearing, or whether they were systemic cases, in which the tonsils were operated upon as foci of infection. In the second class of cases, while he had no detailed statistics at hand, results had encouraged him to continue to take out tonsils in the presence of such indications. There might be other indications for removing the tonsils, laryngeal tuberculosis, for example, had been known to improve after removal of the tonsils. In one case which he recalled the vocal cords were a mass of infiltration, suggesting the necessity of laryngectomy. The patient was sent to a lung specialist, a section was removed, and typical tubercles found. The tonsils were removed as a prophylactic measure, and the patient recovered entirely. The tonsils had also been suggested as the foci of infection in poliomyelitis.

**Dr. George F. Cott**, Buffalo, N. Y., recalled two cases of systemic diseases in which removal of the tonsils was resorted to. In one, a woman with arthritis of mild character, in which she could not straighten her left knee for six weeks, was able the next day to do so without pain, she left the hospital in less than a week. In another woman, aged 44 years, with a large goitre and arthritis, after removal of the tonsils her symptoms were much ameliorated and two-thirds of the goitre disappeared.

He recalled a case in the practice of a colleague who did a minor uterine operation on a patient with goitre, intending to remove the goitre later; the patient promptly died.

The speaker presented lantern slides showing lung abscess. Artificial pneumothorax was doubtless the most efficient treatment. It should be employed by the laryngologist. The reader might be easily instructed, and without danger to the patient, by using the fluoroscope.

**Dr. Wolff Freudenthal**, New York City, unlike Dr. Richardson, sometimes had vomiting during operation. In view of the lymphatics opened in the course of operations on the upper air passages it was remarkable that more cases of infection were not encountered. Six out of eight cases cited by Manges had occurred in one hospital in New York City. This was significant.

He did not agree with Dr. Layman with reference to prophylactic tonsillectomy in every child.

**Dr. Lee Cohen,** Baltimore, Md., emphasized the importance of formulating some general procedure in operating in the presence of focal infection. There were various focal infections which were manifest in the absence of disease of the tonsils. Among a number of cases referred to him, in one there were definite deposits in the joints, and in one there was definite neuro-retinitis. He made cultures to ascertain whether organisms could be found. In twelve cases the cultures showed streptococcus viridans associated with pneumococcus. In these cases the results were absolute. The internist had excluded every other source of infection before the cases were sent to him.

**Dr. Robert H. Craig,** Montreal, Canada, had had one case of lung abscess attributable to the anesthetic. Since this occurrence, in operating upon adults he had preferred to use intratracheal insufflation, for which he claimed many advantages. The operation was facilitated by having the anesthetist out of the field of operation. A continuous flow of ether and air, warmed through a water-bath to body temperature, was administered in any mixture of the two, from pure air or oxygen to pure ether, the average being 35 per cent. ether to 65 per cent. air. The ether-air vapor was forced into the lungs through the tracheal tube with five to ten millimeters mercury pressure for children, and ten to twenty for adults, thus insuring a forced exit of air from the glottis, which prevented the aspiration of mucus, blood or other oral or nasal secretions. The operation could be done more deliberately, as once the patient was completely anesthetized the amount of anesthesia could be more carefully gauged than by any other method of administration.

The recovery from the anesthetic was accomplished almost without shock or vomiting, as the patient could be given air or oxygen through the tracheal tube after the operation was completed. It was necessary to have an expert anesthetist for this work.

**Dr. Layman,** in closing the discussion, did not wish it to be inferred, as Dr. Freudenthal had suggested, that he advocated tonsillectomy in all cases.

## EXAMINATION OF THE LARYNX AND UPPER END OF THE ESOPHAGUS WITH THE HEAD STRAIGHT.

By RICHARD HALL JOHNSTON, M. D., Baltimore, Md.

*Technique.* Supine Position. After wrapping and pinning the child in a sheet so that arms and legs are practically immovable, he is placed supinely on the table with the head straight and in the same plane as the body. An assistant stands at the end of the table and steadies the head between the hands. A nurse attends to the body and limbs. Standing to the left or right side of the table, the operator passes the 10 mm. laryngoscope—long for older children, short for infants—between the respective bicuspid teeth, pushes the tongue to the opposite side, raises the epiglottis and exposes the larynx in a few seconds. If the operator is right-handed, it is better to hold the tube in the left hand and pass it from the left side of the table leaving the right hand free for operative procedure. In the case of a left-handed operator the right side of the table must be used. After viewing the larynx, if it is desirable to examine the upper end of the esophagus, the spatula end of the instrument is pushed back of the larynx and, usually without a lifting movement, the esophagus opens up and can be quickly observed for strictures or foreign bodies. While not absolutely necessary, a full dose of atropine a half hour beforehand will aid the operator in securing a dry throat. The exposure of the larynx and upper end of the esophagus is done so quickly that it is not necessary to starve the patient.

The above technique is used in adults under general anesthesia for operating in the larynx and for the examination of and the removal of foreign bodies from the upper end of the esophagus. It is rarely the case that general anesthesia is required in direct laryngoscopy in adults. They can be examined and treated in the sitting position with the head straight or slightly extended if the 10 mm. tube be used. In such cases the head is supported between the palms of the hands of the nurse.

*Anesthesia in Direct Laryngoscopy.* In children up to ten years of age, no anesthesia, local or general, is necessary in routine work. After ten years the operator must use his judgment as to anesthesia. In one patient, eleven years old, the writer did

an esophagoscopy under cocain with the boy in the sitting position: in a boy, fifteen years old, a papilloma of the trachea was removed through the 9 mm. bronchoscope under local anesthesia in the sitting position. In adults the writer prefers alypin anesthesia—20 per cent. solution which can be used ad libitum though, in most patients, a small quantity suffices. In operative cases a hypodermic of morphin, $^1/_8$ to $^1/_4$ of a grain and atropine, 1/150 of a grain helps greatly. In foreign bodies in the bronchi in adults in which anesthesia is necessary, the bronchoscope is easily and quickly passed through the direct laryngoscope with the head straight: the head is then lowered to the plane of the table, the laryngoscope removed and the bronchoscopy proceeded with.

*Instruments used.* With three tubes one can examine all patients. For routine work the writer uses the modified Jackson 10 mm. tube which exposes the larynx and upper end of the esophagus in adults and older children. This tube can also be used in infants but is somewhat clumsy in very small throats. For the latter class of patients the writer uses a much shorter separable tube which is valuable in passing the 4 mm. bronchoscope and in the removal of flat foreign bodies such as watermelon seed from the trachea of the infant. In three such cases the writer has introduced the baby speculum with the head straight and has removed the foreign body by passing forceps between the vocal cords without the use of a tracheoscope. The third tube which is useful in operating in the larynges of adults is the Jackson 12 mm. laryngoscope, the largest the writer uses in his operative work.

*Advantages of the straight method.* The greatest advantage is the fact that no trained assistants are needed. One can operate at the house of the patient with the aid of the family physician and the mother or child's nurse or at a hospital in which such work has not been seen. When working in private homes it is not necessary to give atropine or to make any preparation. More than once the writer has made examinations as above described and in one patient —a boy, ten years old.—who had swallowed a collar button which lodged in the larynx and who had been tracheotomized by the family physician to prevent suffocation, the writer traveled ten miles in the country at night to remove the foreign body with the head straight. The assistants were the family physician and the father, neither of who had seen a direct laryngoscope. No anesthetic was used. In another patient, four months old, a baby pin was removed from the larynx in the office of Dr. W. S. Baer a few minutes after the X-ray

picture had revealed its presence. Again the assistants were men who had never seen the instrument used.

Recently the writer removed an open safety pin from the larynx of a baby, six months old, who was brought to his office on Sunday after hours by the family physician who, with the child's nurse, rendered first aid.

Another child, fourteen months old, with a penny at the upper end of the esophagus was brought to the office from the country and was quickly relieved of its burden with the help of the family physician and the father. Since this was written the same physician has brought another patient, eighteen months old, with an overcoat button at the upper end of the esophogus. This was likewise quickly removed in the office with the head straight. One Sunday night a physician called the writer and asked him if he would see a baby immediately with some obstruction in the throat. The history was that the child, one month old, has had, since birth, attacks of difficult breathing and cyanosis. When the child was brought to the office about 10 o'clock, the face was slightly blue. The examination was made at once and showed a normal larynx. The next day Dr. H. E. Ashbury took X-ray pictures which revealed an enlarged thymus gland which gradually yielded to treatment.

These cases are not cited to advertise the skill of the writer but to emphasize the ease with which straight direct laryngoscopy and esophagoscopy can be done. It is so simple that it puts direct laryngoscopy within the reach of every laryngologist. The instruments needed are few; hospital team work and trained assistants are not needed for successful work, and the head of the patient lies before one in the normal natural position instead of "upside down" as it appears to some beginners in extension. The landmarks are more easily recognized because there is no possibility of their getting out of the normal position. After the exposure of the larynx, the hand holds the instrument in position without strain as long as may be necessary. In two cases of papillomata in children—a boy, ten years old, and a girl of five years—under general anesthesia the writer exposed the larynx fifteen minutes and applied radium between the vocal cords with the right hand. One point in the removal of foreign bodies from the upper end of the esophagus is of importance. With the head straight the 10 mm. tube approaches the esophagus at an acute angle which makes it impossible to override the foreign body.

Flat objects sometimes lie in the posterior plane of the esophagus and the tube, passed straight, may fail to reveal the intruder. The writer has never failed to find the foreign body at the first examination with the head straight.

In conclusion it may be in order to cite an incident which emphasizes the importance of having a working knowledge of direct laryngoscopy. About four years ago the writer was asked to examine with the tube the throat of a girl, six years old, who had been hoarse eleven months. Tonsils and adenoids had been removed by the family physician without benefit. In March the little patient was taken to a specialist in Baltimore, Professor of Laryngology in a medical school. He was unable to see the larynx with a mirror but had made a diagnosis of multiple papillomata and had treated the condition with sprays and gargles. In July, when no benefit had come from five months' treatment, the parents insisted that something further be done. The writer was asked to pass the laryngoscope at the hospital with which the Professor was connected. He was present at the examination and talked confidently of finding the larynx filled with papillomata. The patient was examined without anesthesia with the head straight. When the larynx was exposed a dark mass was seen in the subglottic space which proved to be a foreign body. The Professor was greatly chagrined when no papillomata were found. This story carries a moral. If laryngologists do not want to make false diagnoses or to take the risk of being shown up by a fellow specialist, innocently or intentionally, they had better take the trouble to learn to see the larynges of all children by simple, straight, direct laryngoscopy.

# A NEW METHOD OF WORKING OUT DIFFICULT MECHANICAL PROBLEMS OF BRONCHOSCOPIC FOREIGN-BODY EXTRACTION.

By CHEVALIER JACKSON, M. D., Philadelphia, Pa.

The endoscopic removal of a foreign body that has no sharp edges or points to catch in the bronchial wall and that is not impacted is quite simple. On the other hand, certain foreign bodies with long and sharp points or with acute angles may require much ingenuity to disengage them so that they become free to move and safe to make traction upon with forceps. To pull upon an entangled foreign body in either the bronchi or esophagus is to court disaster. If such traction be not fatal, as it usually is, ultimate removal is made much more difficult or impossible.

In this disentangling procedure when all previously made instruments had failed, the author found it possible to solve the problem by means of probes and various forms of instruments bent and made to suit the purpose demanded by the mechanical problem presented in the particular case. In some cases it is possible to work out beforehand a curve or an angle that will do the work; in other cases it is necessary patiently to form and reform the end many times, testing each time, until the precise angle or turn or series of angles or turns is obtained to do the work.

The faults of stock instruments for this purpose are two: 1. They may be of not quite the proper shape. 2. They may have been subjected to too sharp bending which destroys the tensile strength of the steel. 3. If the shape is not exactly proper the instrument will not accomplish its purpose.

If the tensile strength has been impaired the instrument may break; because endoscopic instruments are necessarily delicate and the maximum tensile strength of the best tool steel (Stubb's) is none too great when reduced to the thread-like size needed for work in solving a difficult mechanical problem in the small bronchi of children. When the endoscopist himself works out an instrument he knows that it has not been subjected to strains of strength-impairing character. If such strains occur he should throw the steel away and start afresh.

The author's procedure when a difficult case is encountered, is to study over his previous cases of the same kind of foreign body; if, as is now usually the case, he has had to deal with similar foreign bodies. Next, a bronchoscopy or an esophagoscopy, as the case may be, is done, with all previously used instruments at hand for use if, on endoscopic study, they meet the requirements of the problem. If they do not, mental notes and measurements are taken and a series of probes or other instruments is made. These are tried out thoroughly on the work bench using a piece of rubber tubing for a manikin. One end of the tubing is anchored in a small vise which lies on the table, giving stability to the distal end of the tubing (Fig. 1). If the instruments will not enable the endoscopist to disentangle the foreign body in this manikin, obviously they will not enable him to do it in the living patient;

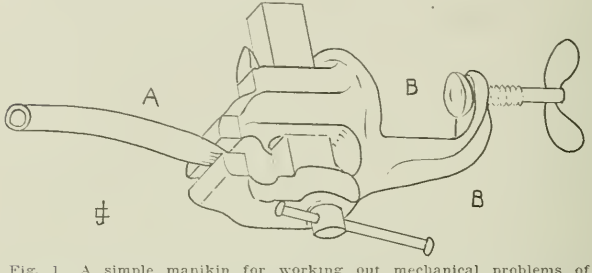
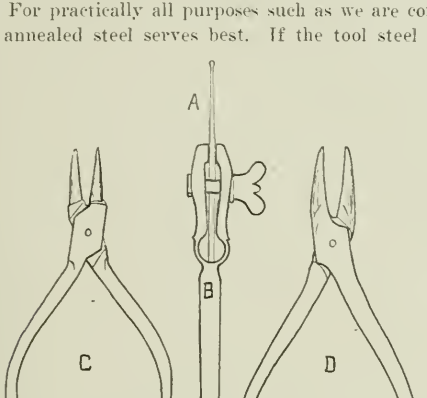



Fig. 1. A simple manikin for working out mechanical problems of foreign body extraction. Almost any problem can be simulated. The vise lies flat upon the table and the size of tubing is selected to correspond to the size of the bronchus.

and a continuation of careful, patient study is undertaken until the problem is solved.

Having solved the simulated problem on the manikin, the solution of the real problem in the patient is attempted, and usually with success. If unsuccessful a slight modification of the probes may succeed. This modification is made by means of the spirit lamp and the pliers (Figs. 2 and 3), which are sterile and ready on a sterile table in the operating room. The instrument must be allowed to cool slowly. If chilled quickly, as by immersion in water, the steel will be hardened and consequently made brittle. Cooling slowly anneals the steel. These slender instruments cool almost instantly. They should be, when finished, withdrawn slowly from the flame in an upward direction so that the heat after leaving the flame

will prevent sudden chilling in the air. If some degree of spring is desired the finished instrument can be hardened "right out" by immersion in water when dull cherry red. Then the temper can be drawn to the degree desired by any one of a number of methods. This tempering had best not be attempted by any one not thoroughly familiar with the procedure. Fortunately, tempering is very rarely required. For practically all purposes such as we are considering here, the annealed steel serves best. If the tool steel is of the




Figs. 2, 3 and 4. Pliers for the bending of probes. The surfaces of the jaws are polished so as not to roughen or mutilate the probes in bending. At A is shown a probe-shaped steel rod, a number of which are prepared beforehand for shaping to suit the particular mechanical problem when encountered. The probe is shown held in a jeweller's hand vise for shaping. After removal from the vise a handle is made by bending over to a right angle about 1 cm. of the proximal end.

highest quality it will have sufficient stiffness when annealed. Under no circumstances should work of this kind be attempted with wire, machinery steel, or iron.

On the tool table, in addition to the newly-wrought instruments, are a number of straight steel rods formed with probe-shaped ends (Fig 4), and a number of straight unwrought rods. These are for forming an entirely new instrument if desired, though this will rarely be necessary unless the position of the foreign body has shifted, thus changing the mechanical problem. Such shifting rarely occurs in the class of cases here considered.

The probes (Fig. 4), are made by filing in a watchmaker's lathe. Of course they can be made up in quantities at the instrument maker's, but those made from the steel rod by the endoscopist himself inspire the most confidence. They can be filed to shape while being held in a hand-vise, if no small lathe is at hand; but the lathe-made probes are more quickly and conveniently wrought to shape. In the solution of some problems a flattened or a squared cross section on the end of the probes is advantageous. In this case the cone of the lathe is locked at the proper position and accurate filing can be done. The most convenient form of handle, it has seemed to the author, is the simple right-angle bend shown in Fig. 4). This is always made last and is turned so as to be opposite the bend formed on the distal end; so as to be in all instruments in the same relation. By this means the position of the probe-end is always in mind.

In forming probes for the purposes herein mentioned it is necessary to see that they are not hooks; that is, that they do not approach too closely to a right angle. A more or less obtuse angle or an approach to spiral form will always permit them to be withdrawn by backward rotation. To have an instrument so entangled as not to be able to disengage it would be a serious or possibly fatal predicament, as reported by Hubbard in the use of a snare.

Great caution is necessary in the use of these bent probes and the utmost delicacy of the sense of touch must be exercised, as it is very easy to perforate the bronchial wall with either the probe or the foreign body.

### *Tools.*

The tools needed for this work are mainly those of the jeweller, not those of the blacksmith or machinist. Extreme delicacy and precision are essential. Lack of these will result in either a clumsy instrument or one with weak places.

*Lathe.* A good precision lathe is essential, and it must be delicate. The Webster-Whitcomb jeweller's lathe is a standard quality. It should have a full assortment of chucks and attachments. In passing it may be said that the mastoid curets of the otologist can be sharpened from the inside of the bowl by a carborundum bar held in this lathe, so as to cut in a way that will seem incomprehensible to those used to dull curets.

*Work-bench.* A firm, solid work-bench at a window where good bright daylight may be had is essential.

*Vise.* A small jeweller's bench-vise is needed. It should have detachable leather facings for the jaws so as not to roughen the probes when they are held after finishing. In the rougher part of the work the more accurate holding of the bare jaws is needed. The common cast-iron vise is a mere toy and will not serve. A small hand-vise or a jeweller's pin-vise is also needed.

*Files.* A set of jeweller's needle files is best for this delicate work. They are made of twelve different forms of cross section.

*Pliers.* Square-nosed, round-nosed and pointed flat-nosed pliers are needed. Two sets are essential; one set with roughened jaws for the roughing-out, and one set with absolutely smooth and polished jaws for bending the finished probes. The pliers used by opticians for fitting spectacle frames are best for the latter purpose. A pair of heavy cutting pliers is convenient for cutting off the probes, or other instruments, to length.

*Anvil.* A jeweller's anvil is very convenient as is also a bench-block of faced and smoothed cast-iron, for straightening rods, etc.

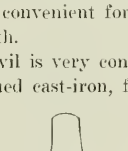


Fig. 5. Glass tube used for demonstration of mechanical problems and sometimes for study of them.

*Polishing tools.* A good jeweller's motor with carborundum grinding wheel and a buffing wheel is very convenient. Emery cloth of finest grain, and rouge cloth are needed for hand-smoothing and lathe-finishing. A high polish is objectionable, but the instruments should be smooth. They may be blued after finishing by heating in the bunsen flame. The dark blue instruments of dull finish are more easily seen.

*Alcohol lamp* is convenient because it can be sterilized and placed on the tool table in the operating room for bending probes. For use in the work shop the bunsen gas burner may be used.

*Manikin.* The manikin of Killian was made to teach introduction of the tubes. It was for use in the sitting position. The manikin of Von Schrötter was for teaching introduction in the lateral position. Hubbard has made an excellent manikin for use in

simulating the recumbent position. The author uses for the working out of mechanical problems a bit of drainage tubing about 20 centimeters long, the distal end being held in a small cast-iron vise that can be purchased for twenty-five cents at any hardware shop. In use it is not fastened to the bench, but allowed to rest on the

Fig. 6. Schema showing how a bar pin was pushed down by ill-advised alternate pushing and pulling until it had been forced into the lowest possible position. Complete perforations as shown at B and D are usually fatal.

bench, desk or table. Much practice may be had with it at odd moments. For demonstration of mechanical problems, and sometimes for study of them, a glass tube is used, Fig. 5.

*Other tools.* The other tools that are convenient and that the mechanically-inclined endoscopist can and will naturally add to his

workshop are too numerous to mention. Saws, drills, reamers, screw-plates, taps, dies and so on *ad infinitum* can be used with pleasure and profit by those who naturally turn to such work as recreation. But it is not for such that this article is written. It will not pay the busy bronchoscopist to make his own instruments for general work. There are some instruments, however, such as those herein before described that he can make for the particular case that will enable him to get out a foreign body that otherwise he will never get out.

*Illustrative Case.*

Infant, aged 4 months, aspirated a bar pin, to remove which two attempts had been made, first under ether anesthesia, next under chloroform. Tracheot-

Fig. 7. Schema illustrating a new method of closing a bar-pin.

omy had been done as stated by my predecessor in the case (who has since urged me to make this publication of it) for the combined reasons that (a) the first bronchoscopy was followed by subglottic edema; (b) the pin was supposed to be in the cervical trachea reachable by tracheotomy; (c) this failing, removal could be accomplished by bronchoscopy through the tracheotomic wound. These procedures all failed however and the child was brought to our clinic. Temperature 104.3°, pulse 158, respirations 52. The breath was foul, the lips were cut, sloughing shreds of mucosa were visible in the pharynx. Mucopus was streaming down the neck from the tracheotomic wound in which was an over-sized aluminum cannula full of crusted secretions. The child was pale and very fretful. It moaned almost constantly, and slept but little. We did not expect it to live longer than a few hours; and in consulta-

tion with my predecessor in the case it was decided that nothing was to be done but to try to get the child to rally. It was clear that it was suffering from surgical shock and sepsis, not from the pin. Contrary to our expectations the child lingered along for a few days and then slowly began to improve. At the end of three weeks the temperature was almost normal and the child was in fairly good condition. There was a loose cough, dullness over the left lower lobe, which was full of rales. A radiograph showed imperfect drainage

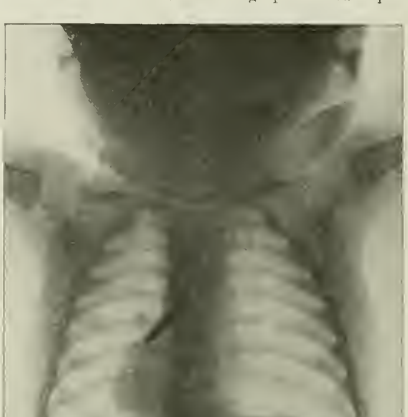


Fig. 8. Radiograph showing bar pin in left bronchus of 4 months old infant.

Fig. 8 (b). Bar pin removed bronchoscopically from the left bronchus of an infant of 4 months.

from the left lower lobe. The bar pin was seen to be in the left bronchus about six millimeters below the bifurcation.

Without anesthesia, the 4-millimeter bronchoscope was passed through the mouth. There were patches of exudate in streaks on the tracheal wall. Pus filled the left bronchus, and no air seemed to be passing in or out of the left

bronchus, whose walls were so swollen that the end of the pin was hidden from view. The pin was apparently wedged fast. It was thought best not to make any attempt to work with it for a time, for both local and general reasons. Meanwhile a method of removal was to be worked out in the bronchoscopic laboratory.

It was quite clear from the statements of my predecessor and from endoscopic inspection of the case that what happened was this: As shown schematically in Fig. 6, the bar pin had been at first lying loosely in the trachea, as shown at A. Traction resulted in causing the pin-point to enter the tracheal wall as shown at B, preventing withdrawal. To liberate the point, the pin had been pushed downward to a new position (c) where traction again caused the point to enter the bronchial wall. This had been repeated until the pin was forced down tightly as far as it could go in the small stem bronchus of a four months old infant, and there anchored with what seemed utterly hopeless tightness. Of course some such ratchet-like action downward occurs from cough and even respiration; but my predecessor nobly took the blame upon himself, claiming that the intruder if unmolested could not have arrived at such a condition of impaction and anchorage. The greatest difficulty arose from the fact that the forcing in of the pin together with the edema left no

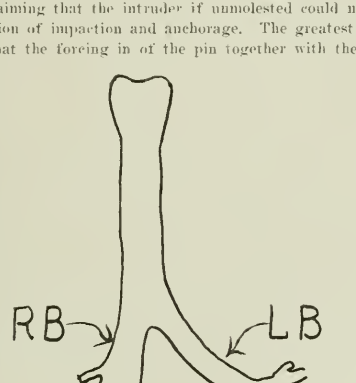



Fig. 9. Cast of the trachea, main bronchi and stem bronchi of an infant aged one year. Actual size. Note size of main bronchi. They are not larger than a large straw.

room at the side of the pin to work. Any instrument inserted would need to make room for itself by displacing the edema and the utmost care would be necessary to prevent perforation of the bronchial wall by the instrument or the foreign body. In addition to this it should be remembered that the stem bronchus of a child of this age is not over three millimeters in diameter (Fig. 9). It seemed as though bronchoscopy had once more encountered the imposi

ble. To pull the intruder out at all hazards would certainly have been promptly fatal, and was not to be considered for a moment.

The method finally worked out on the manikin is shown schematically in Fig. 7. The fine, bent probe, B, was formed to the proper curve to be insinuated by rotation to the position shown. Then by gently turning the bent probe, at the same time making gentle pressure with the fixing push rod, C, the pin was closed. This rendered forceps withdrawal safe and not difficult. No anesthetic general or local was used. The child made a good recovery and has remained entirely well.

Part of the inconceivable difficulty here presented was in the small size of the trachea and bronchi in an infant of four months, coupled with the limited time that it is safe to continue working.

The main bronchi are not larger than a goose-quill and to stretch them by forcing in a large tube is certain death. The lumen diameter at four months of age is not more than half what it is at twelve months.

My predecessor in this case asked that mention of his name be omitted in order that I might feel absolutely unhampered in fully stating the case so that others might avoid committing the error of pursuing a foreign body downward.

For use in the esophagus, forceps may be substituted for the fixing rod.

## *Conclusions.*

1. Tool-steel probes formed by the bronchoscopist himself, to suit the particular case, are an addition to our means of solving mechanical problems of bronchoscopic foreign-body extraction.

2. The form herewith shown can be used to close a bar pin, which cannot be done with any of the different forms of safety-pin closers.

3. To pursue a foreign body downward is to court disaster.

4. Care is necessary to avoid forming hooks that could catch in bronchi or become entangled in the foreign body. The shape should be such as to favor "unscrewing" out if caught.

5. Great care should be taken to avoid injury to the patient. Any number of trials may be had if no harm is done. One serious trauma may terminate the case.

# FURTHER OBSERVATIONS ON TRACHEOBRONCHIAL DIPHTHERIA, WITH REPORT OF ADDITIONAL CASES.

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Since my last report on this subject* diphtheria cases have not been prevalent in New York City, and during the summer months of 1916, for the first time in the history of the institution, there were no intubated cases admitted to the Willard Parker Hospital, the severe epidemic of poliomyelitis apparently having taken the place of diphtheria and the exanthemata. During the year preceding there was also a small percentage of tracheobronchial diphtheria even though there were many admissions to the hospital. All of the cases were mild and many of the "croup" cases recovered without intubation after a large dose of antitoxin. The writer has stated in former papers on this subject that he does not consider a case tracheobronchial in whom the intubation tube of Dr. O'Dwyer relieves the stenosis. It is only those cases with membrane below the tube causing obstruction to respiration that he considers tracheobronchial clinically and which call for bronchoscopic measures to relieve the obstruction. There were, however, a few interesting border line cases with diminished respiratory murmur and increased resonance over the affected lung. One of these cases had an encapsulated empyema that had ruptured into the superior lobe bronchus which Dr. Ferguson of the attending staff had aspirated by needle with negative results. It was completely cured by aspiration bronchoscopically, a dram of pus showing pneumococci being removed. Another case, an infant with markedly bulging right chest wall in comparison to the left chest, which was apparently collapsed, and who was in extremis and died five minutes after admission, was found by postmortem bronchoscopy to have an impacted hull of a pistachio nut imbedded in the right bronchus. Another case had a safety pin in the right bronchus. It was not diphtheria, and the patient recovered after the removal of the pin. Still another case was admitted with the history of having aspirated a foreign body (a piece of egg shell) which proved to be tracheal diphtheria. We are constantly on watch for tracheobronchial cases. Dr. Gover

*See Laryngoscope, October, 1916.

of the resident staff examines all of the cases showing respiratory difficulty by direct means and he feels that he can practically foretell from the appearance of the larynx and subglottic edema the cases that require intubation and the cases that do not require it. He has also gathered some very valuable data about the condition of the larynx as well as the physical findings in these cases and after further study will make a report on this subject.

There were also admitted some very interesting cases with bilateral bronchopneumonia intubated before admission for supposed laryngeal and tracheobronchial diphtheria and reported as such; these proved on examination by direct inspection to have no involvement of the larynx, trachea or bronchi, and when detubated promptly remained without the tube. I am truly happy that Dr. Gover has taken such an interest in the study of the cases, for it is very essential to have careful data on these cases in order to prove which are diphtheritic from the presence of clinical evidence of membrane, and which are mistaken for diphtheria and wrongly diagnosed. We have seen no untoward effect on any of these children suffering with pneumonia, and after bronchoscopy and suction the child was apparently breathing much more comfortably than before the examination.

In the selection of the bronchoscopic tubes for examination we have adhered to an old rule of the writer to use the smallest size tube possible when performing intubation and not be governed by the age of the patient in the selection of the tubes. Therefore, the bronchoscopic tubes most frequently used on these children are the 4 and 5 mm. tubes of Dr. Jackson. By using the small tubes one makes little traumatism and at the same time trains the eye to see and interpret objects through a small lumen which is the main essential of all bronchoscopic examination. With the larger tubes objects are easily recognized but this is not the case except by constant practice in the living with the 4 mm. tube that distant objects are discernable.

The duration of time should be taken into careful consideration. It should be the least possible duration with an endeavor to relieve the primary diphtheritic obstruction. The duration of time is extremely important as the acute diphtheritic obstruction must be removed at the first trial and if the desired result is not accomplished later bronchoscopy does not aid in the recovery of the case. So far as I have been able to judge in my limited experience with bronchoscopy aspirated food and peanut pulp are equally as irri-

tating and fatal if not removed at the first trial. The average duration by actual time in minutes has been from five to eight minutes in children. The bronchoscopic tube may, however, be left in situ with no apparent discomfort to the patient for a period of fifteen minutes after spraying or swabbing with antitoxin. The children breathe with perfect ease and often fall asleep with the bronchoscope in situ after the obstruction is relieved. In adults the duration may be prolonged but the general condition of the patient should be taken into consideration as well as the amount of involvement of the bronchus.

In the removal of the membrane by suction or forceps great care should be taken not to cover the attached portion of the cast with the lip of the bronchoscope but to locate the uppermost part of the attachment of the membrane and follow it down to its distal attachment. I have had this difficulty arise where I could see the flapping piece of membrane which, being held to the tracheal wall by the tube, was difficult to detach *en masse*. The membrane ordinarily is stripped off from the tracheal and bronchial wall readily but in the instance I mention I had some difficulty by overriding the proximal attachment of the membrane by the lip of the tube.

In compiling statistics on these cases I have recorded all of the cases handled by the house staff or myself even though they succumbed within a period of from twelve to twenty-four hours after admission. This, of course, greatly increases the mortality rate, but it is only fair to report the good results as well as the bad and then deduct the twelve to twenty-four hour cases showing what may have happened had the cases not been of such long duration had they been recognized early.

Of the case reports I have eleven to add during the years 1915 and 1916. One adult was admitted to the Willard Parker Hospital with tracheobronchial diphtheria who recovered. All of the others were children from one to eight years of age. Of the eleven cases treated six recovered and were discharged cured. Five died, three of them being in moribund condition on admission. One died one hour after admission, another within twelve hours, and a third in seventeen hours. The other two died from extensive pneumonia fifteen and twenty days after the removal of the membrane. The pneumonia developed in one case after the temperature had reached normal on the seventh day and the child was without the tube. Deducting the cases that died within the first twenty-four hours would make a very encouraging re-

covery rate 75 per cent., but we do not deduct any case in whom intubation has been performed or in whom bronchoscopic examination has been made, and therefore the high mortality rate with such cases inclusive is rather discouraging. Following is a brief report of the cases:

*Case 1.* A. W. A woman 35 years old, ill five days, was admitted to the Willard Parker Hospital January 10, 1915, suffering with laryngotracheal diphtheria. On admission the woman was in poor condition, markedly cyanotic and suffering from extreme dyspnea. She was promptly intubated with an adult intubation tube by Dr. Archibald Dickson, the Resident Physician, and the stenosis was temporarily relieved. Within a short time the tube became obstructed and was removed and a small cast of membrane followed. As the coughing up of the cast apparently relieved the stenosis, reintubation was not necessary and the patient breathed fairly well for one hour but gradual stenosis necessitated reintubation. During the interval that she remained without the tube a physical examination of the chest was made and a dose of 40,000 units of antitoxin administered. The physical examination was rather unsatisfactory owing to the very thick chest wall of the patient, who weighed about 230 pounds. However, Dr. Dickson thought there was diminished respiratory murmur over the right lung in comparison to the left and that bronchoscopic examination was indicated. None of the other members of the house staff or myself were able to corroborate the findings of Dr. Dickson on physical examination, but the bronchoscopic examination proved that Dr. Dickson was the only one of us who was correct. The tonsils and pharynx were covered with membrane. The arytenoid cartilages were enormously swollen and the right one covered with a thick exudate. The vocal cords and ventricular bands were beefy red, the right cavity of the larynx being covered with membrane and the left free where the cast had possibly been removed by intubation. The 7 mm. bronchoscope of Dr. Jackson was introduced, as the 9 mm. tube would have been too large to pass into the larynx without causing much traumatism. On introduction of the bronchoscope a well defined cast of membrane was visible on the right antero-lateral tracheal wall extending downward. This was promptly removed by suction and the tube passed downward until the carina came into view when the membrane was seen in the right bronchus. This piece of membrane was also removed by aspiration but the suction tube became obstructed and another was substituted. The extension of the cast continued into the superior lobe bronchus and also as far down as the middle lobe orifice but did not enter it. All of the membrane was removed from the superior lobe as far as I could see. The mouth of the bronchus was much swollen. A suction vacuum of ten inches was used. The bronchial area was swabbed as well as possible with antitoxin. While swabbing with antitoxin the nurse did not tighten the sleeve on one of the Coolidge sponge holders and the gauze sponge became detached on removal and we had a foreign body added. The gauze fortunately became lodged in the mouth of the swollen bronchus and was removed. Applications of antitoxin were made to the trachea and larynx and a long 13 cm. intubation tube introduced. A dose of morphia gr. $\frac{1}{4}$ and atropia gr. 1-150 was administered one-half hour before bronchoscopy. No local or general anesthetic was used. Although eocain was not used I do not think it was contraindicated. The duration of

bronchoscopy was thirty minutes and the tube was left in situ for fifteen minutes longer to allow for free breathing and to remove mucus and insufflate oxygen. The patient apparently suffered from little shock and had a fairly comfortable night. The following day the temperature was 103.2° Fahrenheit, pulse 110, respiration 38. The tube was removed four days later and on removal of the tube through the direct speculum a mass of curdy digested membrane was coughed out. The larynx was free from membrane but considerable ulceration remained. No subglottic edema was noted. The tube was not replaced. The temperature gradually reached normal on the tenth day and the patient was discharged from the hospital one month later. The voice was very hoarse at the time of discharge from the hospital due to an infiltrated and fixed cricoarytenoid joint on the right side. The patient insisted on leaving the hospital and going to Atlantic City even though we advised her to remain in the city for fear of return of the stenosis. Dr. Dickson saw her three months later and reported that the arytenoid was moving and the voice was returning to normal. There were no complications. All of the cultures were positive.

*Case 2.* W. J., 8 years of age, ill five days, was admitted to Riverside Hospital January 28, 1915, for postnasal, glandular and laryngeal diphtheria. The ambulance surgeon who brought the boy to the hospital did not consider intubation necessary and thought the whole trouble was due to obstructed pharyngeal-croup from the enormous amount of swelling of the neck and face. A careful physical examination was made by Dr. E. M. Goldstein on admission and he noted the supposed enormous collar of brawny cellulitis to be due to subcutaneous emphysema. The neck and face were enormously puffed out with air and the ocular and palpebral conjunctivae of the left eye stood out as a large air bubble. There was but slight exudate on the tonsils but the uvula was gone and was thought to have sloughed off, but a history from the parents the following day cleared up this error. The tonsils had been removed and the uvula was probably removed with them; however, there was not sufficient exudate present to account for such clean amputation of the uvula and part of the posterior pillar of the right side.

There was a peculiar inspiratory-expiratory wheezing accompanied by a cough which was not sufficiently constricted to require intubation. The heart action was rapid but regular. A dose of 10,000 units of antitoxin was administered, 5,000 units having been administered prior to admission by the family physician.

On the 29th, the second day after admission, the emphysema and stenotic symptoms had markedly progressed and intubation was resorted to in order to relieve the condition. Dr. E. M. Goldstein, resident physician of Riverside Hospital, intubated by the O'Dwyer method with an 8-9 tube. The intubation instead of relieving the stenosis made it much worse and the patient was almost asphyxiated. The tube was at once removed and following it a cast of membrane was coughed up. He notified me of the condition and on arrival at the hospital I made a thorough physical examination of the patient. The boy was terribly puffed with emphysema and his breathing was decidedly asthmatic. The emphysema was found to extend over the entire trunk down to Poupart's ligament. The arms and hands were also ballooned with air. The pharynx showed a thin exudate on the tonsils, the uvula was gone. The same exudate was in the laryngo-pharynx. A direct laryngeal examination

showed the epiglottis to be free of exudate, nor was there any evidence on the aryepiglottic folds. The arytenoids were enormously swollen but free from exudate. The ventricular bands and vocal cords were plainly visible, free from membrane, but congested. There was apparently marked subglottic infiltration.

A 5 mm. bronchoscope was introduced and small patches of membrane and ulceration were seen below the cricoid extending down into the right main bronchus. Both bronchi were examined but no casts of membrane were seen and the exfoliated cast which had been coughed up was unquestionably the cause of respiratory difficulty. Re-intubation was not necessary.

The case is interesting from the standpoint of subcutaneous emphysema occurring in a case prior to instrumentation of any kind and I was at a loss to explain the cause of the emphysema. The boy had a rather slow convalescence due to myocardial changes and post-palatine paralysis but eventually recovered. The cultures taken were all positive.

At an autopsy which I was able to obtain in a foreign body case other than diphtheria with similar symptoms and subcutaneous emphysema I endeavored to give an explanation of the cause of such subcutaneous emphysema without instrumentation which was as follows: Rupture of the bleb through the pleura into the mediastinal spaces which in turn follows the deep cervical fasciae, and gets out into the subcutaneous cellular tissues of the body was the only plausible explanation for this condition. (See The Laryngoscope, August, 1915, p. 574.)

*Case 3.* M. W., age 20 months, ill three days, was admitted to the Kingston Avenue Hospital, May 16, 1915, intubated for laryngeal diphtheria. The child was in extremis on admission and the tube was obstructed. Dr. Eberle removed the intubation tube but as there was no expiratory cough and it was very evident that the child was about dead, Dr. Eberle rapidly performed tracheotomy and removed a cast of membrane which had caused asphyxia. By means of artificial respiration the child began to breathe but this was followed by a series of convulsions and the baby succumbed one hour later. Although the heart continued to beat during the respiratory failure the profound carbonic acid poisoning was not overcome. Dr. Eberle performed a necropsy and found both bronchi obstructed by membrane.

*Case 4.* T. H., a boy of three years, ill three days, was admitted to the Kingston Avenue Hospital October 3, 1915, intubated for laryngeal diphtheria. On admission the child was in fairly good condition though there was some difficulty with respiration at times and a persistent cough. As the ambulance surgeon said there was immediate relief of dyspnea after intubation, which was apparent on admission. Dr. Thos. Joyce thought the persistent cough was due to the presence of the tube in the larynx to which the larynx had not become tolerant. There was no membrane visible on the tonsils or pharynx. A dose of 20,000 units of antitoxin was given. Two hours after admission the child became very restless and cyanotic and Dr. Joyce, who was hurriedly sent for, removed the tube and found it plugged with membrane. A longer tube was immediately reintroduced which gave the desired relief and the child on return to his crib went to sleep. Within an hour the tube again became obstructed and was again removed and found to be full of membrane. As I was in the hospital at this time Dr. Joyce called me to see the child who, after the third intubation, was apparently comfortable but was slightly cyanosed. On examining the lungs little air was heard entering either side and the little

patient was apparently breathing with great care in order to avoid further trouble. From the history given by Dr. Joyce and the physical findings preparation was made for bronchoscopic examination which seemed to be indicated. The temperature was 102.4 Fahrenheit, pulse weak and rapid and respiration shallow. On removal of the tube the larynx was free from membrane. The 4 mm. tube was passed and immediately ran into a cast of membrane in the trachea; it became obstructed and was promptly removed. The 5 mm. tube was introduced rapidly and also passed into the cast but was withdrawn above the cast, aspiration was applied and a cast was withdrawn hanging to the end of the canula. This was pulled off by one of the nurses and the canula again applied. From the cricoid level downward as far as one could see there was a complete cast of membrane. At times the loose piece would temporarily obstruct vision by flapping into the lip of the tube and covering the light. Two small pieces were coughed through the lumen of the tube and stuck on the operator's mask. It was impossible to remove all of the membrane at one sitting so we decided to discontinue the procedure after we had worked for fifteen minutes and to give another dose of antitoxin, later removing the remainder of the membrane after the antitoxin had had its effect. The bronchoscopic tube was, however, left in situ for fifteen minutes longer to allow for free breathing followed by swabbing and intubation with a long tube. The child apparently was in fairly good condition when he returned to bed and, as he had had 1-32 of a grain of morphia, fell asleep. The same evening the child became very restless and the temperature rose to 105.2° Fahrenheit. There was difficulty with respiration and Dr. Joyce removed the tube. Respiration was much improved on removal of the tube but no membrane was coughed up. Within one and a half hours reintubation was again necessary. The unfortunate little soul was about exhausted by this time and the lungs began to fill with bubbling moist rales. The condition continued to grow worse and the child died from pulmonary edema just twelve hours after admission. No autopsy was permitted.

*Case 5.* W. Z., age 4 years, ill four days, was admitted to the Kingston Avenue Hospital December 19, 1915, intubated for laryngeal diphtheria. There was no relief following intubation for the tube became obstructed and was removed. Further intubation did not improve the condition and Dr. Lauh promptly performed tracheotomy, removing a cast of membrane from the trachea by means of forceps and then introduced a tracheal canula. The patient's condition improved after the removal of the membrane and there was free passage of air through the tracheal canula; 20,000 units of antitoxin was given and the child put in bed. The usual stimulation of camphor in oil was given. The child continued to do fairly well for about twelve hours after tracheotomy when a piece of membrane was coughed into the tracheal tube causing obstruction. The tube was promptly removed by the nurse and a cast of membrane followed. The tracheal canula was reintroduced by the house physician who responded to the call. From this time on the child continued to grow worse and become cyanosed. The heart sounds were weak and rapid and the lungs were gradually filling with moist rales. Repeated doses of adrenalin were given but no amount of hypodermic stimulation had any effect. The unfortunate little boy lingered in this condition for five hours and then passed away just seventeen hours after admission.

*Case 6.* Domonick A., aged 2½ years, ill two days, admitted to the Kingston Avenue Hospital March 23, 1916, was intubated shortly after admission by Dr. Hook, with a 1 year O'Dwyer tube. This tube gave the relief desired and the little patient improved. A dose of 15,000 units of antitoxin was administered. The child had a fairly comfortable night but the following morning he was breathing with difficulty. The tube was removed and was followed by a cast of membrane and much bleeding. Dr. Regan gave a dose of horse serum 30 c.c. intramuscularly and the record states that the hemorrhage promptly ceased. There is not a great deal of bleeding as a rule in these cases where the membrane is attached to ciliated epithelium but there are exceptions however and this was probably one of them. The child was reintubated with a 2-year tube ten minutes later. As reintubation did not seem to relieve the dyspnea Dr. Joyce notified me of the condition and on arrival at the hospital the same afternoon we bronchoscoped the patient.

There was no visible membrane on the tonsils or the pharynx as the speculum was introduced. There was no membrane visible on the epiglottis. The larynx was congested and ulcerated, and the hemorrhage recorded probably came from this region. Ulcerated areas were noticed on the passage of the 4 mm. tube which extended downward into the right main bronchus in which there was a visible membranous cast loosely attached which would flop into the lumen of the bronchoscope with expiration, only to be aspirated into the bronchus with inspiration. With a small suction canula the proximal end of the membrane was readily attached to the tube and a cast of the main bronchus removed with a torn and shredded distal end. The bronchus was then examined for further membrane but none was visible below the upper lobe bronchus though the lip of the bronchus was considerably infiltrated. After the removal of the membrane the usual procedure of swabbing by antitoxin was used and a long tube introduced. On March 26, the tracheobronchial intubation tube was removed but reintubation was necessary four hours later, a two year O'Dwyer tube being introduced by Dr. Joyce. On March 31, the tube was auto-extubated, the same size tube being replaced. The child was in poor condition. There were scattered subcrepitant rales over both lungs posteriorly. The child continued to cough up the tube on an average of one to three times during the day but as soon as a 2-year, non-cough-up tube was introduced the condition was relieved. On April 2, the right side of the face became swollen and on oral examination a small necrotic ulcer (cancrum oris) was noted in the right buccal cavity. Smears from the ulcer showed Vincent's bacillus and spirillum in abundance. The area was treated locally with pure carbolic acid and alcohol, no salvarsan being obtainable. The oral condition continued to progress rapidly so that the necrotic slough was removed by curettage and the area again painted with phenol. The unfortunate little boy continued to grow worse and the area of gangrene increased. The tube was removed for cleansing as there was marked and fetid salivation with a continual flow from the mouth as well as into the lumen of the intubation tube which caused excessive coughing at times. On April 6, a purpuric rash developed on the face and chest, septic in origin. A blood count at this time showed a general count of 15,000 with a polymorphonuclear count of 92 per cent. A blood culture, made with some difficulty by Dr. Eberle owing to the very small veins, showed a hemolysin streptococcus.

The child continued to grow worse and died on April 12, 1916, still wearing the intubation tube. Although the foreign body obstruction was removed early in the disease the many complications which are apt to follow in these cases makes the ultimate outcome extremely discouraging.

*Case 7.* Robert N., age three years, ill two days, was admitted to the Kingston Avenue Hospital April 1, 1917, intubated before admission with a three year O'Dwyer tube. The child was breathing poorly on admission and Dr. Joyce, who admitted the case, also noted on physical examination that little air was entering the right lung. There was no membrane visible on the tonsils. A dose of 20,000 units of antitoxin was administered. As the dyspnea gradually became worse and the child very restless and cyanosed, Dr. Joyce removed the tube. No membrane followed the removal of the tube but the patient was much more comfortable and breathed with little difficulty. It is not at all an uncommon occurrence to have a child breathe easier without the tube especially when the end of the lumen of the tube is covered by a loose piece of membrane. About six hours later, owing to gradual contraction, reintubation was necessary, a longer tube being introduced by Dr. Joyce which seemed to pass the obstruction and give the desired relief. The child remained fairly comfortable with the long intubation tube in situ until the following morning when dyspnea and cyanosis became marked and the tube was promptly removed. With the removal of the tube the distal end was found completely obstructed with thick curdy material, probably digested membrane. The child again remained without the tube and breathed much better after its removal, but later reintubation was necessary, the short three year O'Dwyer tube being used. When I saw the child the same afternoon there were definite signs of obstruction below the tube, viz., prolonged inspiration and shortened expiration, accompanied by the ever present blocked cough. There was diminished respiratory murmur over the upper lobe of the right lung; the left was perfectly free although the respiration was exaggerated. There were few rales. The temperature was 102.4° Fahrenheit, pulse 128, respirations 38 and labored. There was moderate cyanosis.

On direct inspection the tonsils were free from membrane. The epiglottis was not involved but the arytenoid cartilages were red and swollen, which could be accounted for by the frequent intubations. On removal of the tube the larynx was found to be congested and the ventricular bands and vocal cords red and swollen. There was no membrane visible. The 5 mm. tube of Dr. Jackson was introduced which promptly encountered membrane, moving to and fro with every respiratory effort, in the trachea. With the small aspiration tube and a vacuum of from five to ten inches the membrane was removed, the attachment being made at the loose distal extremity, care being taken to see that the cast was not held by the lip of the tube to the tracheal wall. With the suction a cast, 3½ cm. in length, which was part cylinder, a torn cast was removed. The main bronchus and also the upper lobe and stem bronchi were examined, but aside from small areas of ulceration no further membrane was seen. The ulcerated areas were swabbed by the usual antitoxin gauze sponge. A long intubation tube was introduced. The temperature rose following the manipulation which had been by the watch exactly eight minutes, but the child was breathing much more comfortably and soon was asleep. The following day Dr. Joyce reported that there were many scattered rales over the right upper lobe of the lung involved but that the

child was fairly comfortable and taking nourishment well. The temperature remained high, 104.2 Fahrenheit, and the pulse and respiration rapid. On April 5, the long tube was removed by direct detubation and much curdy digested material followed the removal. The child did only fairly well without the tube for thirty minutes when reintubation was necessary, the O'Dwyer tube being introduced. The temperature fell to 101 Fahrenheit the day following the removal of the long tube and reached normal by the end of the week. A second attempt was made to remove the tube by direct means but reintubation was immediately necessary owing to the swollen arytenoids. On April 10, the child was in remarkably good condition but was still unable to remain without the tube. There was not sufficient subglottic infiltration to cause the immediate reintroduction of the tube and therefore the larynx was not cauterized. There was marked adductor spasm, which could be plainly seen, both of the ventricular bands and cords. Such spasms are most frequently the cause of false passages and are even met with today when forcible attempts to intubate are made by the hand of the unskilled intubator. Like all spasms encountered in laryngeal or esophageal tubage the operator who waits for the spasm to relax will do no harm to soft parts, but the over-anxious beginner who is anxious to put his first tube in the larynx, frequently uses such force in the attempt at intubation that he not only ruptures the soft parts and makes a false passage into the mediastinum which invariably has as a sequel to his faulty manipulation a fatal termination from mediastinal pneumonia.

We, therefore, wait for the spasms to relax, especially when the tube is inserted under the direct guidance of the eye, thereby avoiding traumatism and the fatal false passage. Although we had cured the patient of the acute diphtheritic condition we were unable to remove the tube for any length of time. On April 21, the tube was removed and as no immediate spasm followed the child was returned to the crib, breathing well without the tube. This was very encouraging and we thought that at last the child would remain permanently detubated, but such was not the case as Dr. Joyce reintubed him four hours later. We then decided to leave the tube in for one week before disturbing it, for the child had become extremely nervous and frightened at the many attempts at the removal of the tube. On April 28th, the tube was again removed by direct means but the inevitable reintubation had to be done a few hours later. Dr. Joyce, who had given the little child such constant attention, thought that the direct detubation may be the cause of the trouble as the child had become frightened by so many direct examinations and furthermore he argued that even though the removal took not over a minute, the instrument might be the cause of supraglottic edema which I had not seen as I had invariably left the hospital before reintubation was necessary. He therefore suggested that he take the tube out some morning early by the O'Dwyer method and see what happened. He accordingly removed the tube on May 1, and reintubation was not necessary. The child made a perfect recovery. The patient was detained in the hospital for one month after the last removal of the tube to make doubly sure that there would be no recurrent stenosis. He was discharged cured May 31, 1916. This case is interesting from the standpoint of frequent direct examinations being the cause of supraglottic edema as suggested by Dr. Thos. Joyce of the resident staff. No doubt, prolonged direct examination will cause the condition and

does cause supraglottic and arytenoid edema much more often than we suppose; but here is a case with supraglottic and arytenoid edema when examined for not over one minute at any sitting. Most of our cases have been cured after direct methods were resorted to but this case was the interesting exception and was only permanently cured after the skillful digital removal of the tube by Dr. Joyce.

*Case 8.* Martin S., age one year, ill three days, was admitted to the Kingston Avenue Hospital March 28, 1916, suffering from laryngeal diphtheria. The throat was congested but there was no visible exudate on the tonsils. The child was able to cry, showing that there was no involvement of the larynx, but there was marked retraction at times. A dose of 15,000 units of antitoxin was administered, 5,000 having been given just prior to admission to the hospital. The little child had spells of marked dyspnea and eight hours later, on account of the pulse becoming aspirate, that is, disappearing with each inspiration, the child was intubated, a one-year tube being used. After intubation the respiratory difficulty became worse and the tube was promptly removed and a longer tube inserted by Dr. Adam Eberle, which gave temporary relief. Within two hours this tube became obstructed. It was removed and a small piece of membrane followed. The child breathed much better without the tube for over one hour when respiration again became difficult and reintubation was performed with the long one-year tube. On arrival at the hospital a physical examination was made. There was marked retraction even with the tube in situ. Little air was entering the right lung and by inspection the right chest was barrel-shaped. The intubation tube was removed and the 4 mm. bronchoscopic tube of Dr. Jackson introduced. No membrane was visible in the congested larynx, but on introduction of the bronchoscope membrane was plainly visible and very white just at the bifurcation with extension into the right main bronchus.

With a small suction tube the plaque of membrane was removed from the trachea. After the removal of this parietal plaque from the trachea and bronchus further membrane was not seen. The area was swabbed with antitoxin and as the respiratory difficulty was relieved we did not introduce the long one-year tube as is usually the custom after the removal of membrane. However, reintubation was necessary before I left the hospital, a one-year short tube being inserted which separated the subglottic edema and gave prompt relief. There was a sharp reaction following the bronchoscopic manipulation and the temperature rose to 103.8° Fahrenheit. The following day Dr. Eberle thought he could elicit a definite area of pneumonia over the right upper lobe anteriorly which was probably a diphtheritic pneumonia. The pneumonia, however, gradually improved and April 6, the temperature was 100°. From this time on the general condition of the child improved and he made a perfect recovery. The tube was removed and reintroduced six times before the patient finally remained without the tube. The little patient was discharged as cured May 13, 1916.

*Case 9.* Gussie W., age 7 years, ill three days, was admitted to the Kingston Avenue Hospital April, 1916, for laryngeal diphtheria. An attempt to relieve the dyspnea by intubation was performed by the ambulance surgeon at home, but as the tube made the dyspnea much worse he removed the tube and rushed the child to the hospital. On admission Dr. Hook of the Resident Staff intubated the child but there was no relief and the tube became obstructed

with membrane. Further attempts were made to relieve the dyspnea with longer tubes but without success. Dr. Adam Eberle was called and owing to the extreme condition he did not attempt further intubation but promptly performed tracheotomy. On opening the trachea Dr. Eberle removed a cast of membrane and the condition of the child improved. The temperature after the manipulation was taken and found to be 105,2°, respiration 54, pulse 150. Heavy doses of camphor in oil hypodermatically were given, and later, after the child reacted 25,000 units of antitoxin was injected intramuscularly. The child had a fairly comfortable night but became very restless and dyspneic the following morning and Dr. Eberle notified me of the condition. A physical examination before attempting bronchoscopy was of little aid as the constant coughing and rattling of mucus in the tracheal canula rather distorted and magnified sounds elicited by auscultation. However, there was increased percussion resonance over the right upper lobe, the lung which proved later by bronchoscopic examination to be obstructed by foreign body membrane. There was no involvement of the tonsils or pharynx, which were viewed on introduction of the speculum, though there was a slight nasal discharge. As the larynx came into view the tracheal canula was removed. A rapid view of the larynx showed it to be free from membrane but congested from the many intubations. The 5 mm. Jackson bronchoscope was introduced. As Dr. Eberle had removed a cast the night before no membrane was encountered until the carina trachealis was reached when a loosely attached proximal end became visible. This piece of membrane was easily removed by suction on account of its loose attachment and when removed through the tube measured a little over 2½ cm. in length. The cast was a cylinder with a torn and fringed distal end. On further examination of the bronchus it was noted that the mouth of the right superior lobe bronchus was still occluded. This membrane was rather firmly attached and suction only removed very small pieces of it, but with the curved forceps of Dr. Jackson the membrane was grasped and a torn piece 1½ cm. in length was removed. As the child did not stand the manipulation well the bronchial area was rapidly swabbed with antitoxin by means of a Jackson sponge and a long bronchial intubation tube introduced. The tracheal wound was lightly packed with plain gauze and a wet dressing applied. The child was very restless during the night and was given small doses of morphia, 1-32 of a grain every six hours. The next day, April 17, the child was slightly improved and the temperature had fallen to 103°, though cyanosis was marked. On April 18, the bronchial tube was removed. No attempt had been made to close the tracheal fistula which was left open as a safety valve, though it had been loosely packed with a gauze plug and a small adhesive plaster dressing on the neck. There was a profuse discharge from the tracheal wound the following day and the dressings had to be frequently changed. Dyspnea and cyanosis were marked at times and there was almost complete consolidation of the upper lobe of the right lung—a diphtheritic pneumonia. Suction was applied from time to time through the open tracheal fistula and as the child seemed to breathe better without the tracheal canula and owing to the profuse tracheal discharge which constantly plugged the canula it was dispensed with. On April 24, owing to the dried mucopus causing extreme dyspnea and cyanosis, Dr. Joyce reinserted the tracheal canula and by suction removed much of the obstructing secretion. Later he removed the canula only

after it became obstructed and inserted a long intubation tube. Much secretion was coughed through the intubation tube and dyspnea again relieved for the present. Within two hours the intubation tube was removed as it became obstructed and the long cane-shaped tracheal canula of Dr. Jackson once more introduced. The unfortunate little patient lingered in this terrible state with constant obstruction to respiration and frequent changing of the tracheal canula and finally succumbed on April 28, 1916.

This case demonstrates the unfortunate outcome when the diphtheritic membrane is not entirely removed at the first attempt. No doubt the finer bronchi were involved, which accounted for the extensive pneumonia which is in fact pathologically a necrosis of the lung. Such cases are not at all unlike foreign body types other than diphtheria membrane, such as peanut and carrot pulp. These substances, as you all know, are extremely irritating when in the lung and if not removed at the first attempt the case frequently terminates fatally from diffuse bronchopneumonia or abscess of the lung.

*Case* 10. L. W., age five years, ill four days, was admitted to the Kingston Avenue Hospital April 15, 1916, suffering from laryngeal diphtheria and was intubated by Dr. Frick twenty minutes after admission. As the tube gave immediate relief no one suspected that there was membrane below the tube which was to give trouble later. A dose of 10,000 units of antitoxin was administered. The pharnyx was congested but no membrane was seen. There was no nasal discharge. A rather superficial physical examination of the chest which was made showed scattered subcrepitant rales over both lungs. Scattered tubal transmitted subcrepitant rales are not at all uncommon in all intubated cases; no note was made, however, of diminished respiratory murmur.

The following morning at 8.15 o'clock, just twelve hours after admission, the tube became blocked and was immediately extubated and promptly reintubated. There was some difficulty in reintroduction of a 4-5 O'Dwyer tube with which the boy was originally intubated and a tube of one-year size was introduced which gave no relief. Dr. Eberle was called and promptly removed the small tube and reintubated with a 4-5 tube without difficulty. Dr. Eberle notified me of the presence of membrane below the tube and the same afternoon we bronchoscoped the patient. No membrane had been expelled after the frequent extubations and reintubation was immediately necessary. A physical examination by comparison of both lungs showed by auscultation that little air was entering the right lung. The percussion note was higher in pitch over the right upper lobe in comparison to the note over the other portion of the lung, both anteriorly and posteriorly. By direct inspection the intubation tube was removed. The larynx was free from membrane but was very edematous and there had apparently been much trauma in the region of the right ventricular pouch, which probably accounted for the difficult reintubation after the tube had become blocked about twelve hours after admission. The 5 mm. bronchoscope was introduced and membrane was readily seen about 2 cm. below the cricoid level. The membrane was removed by suction, the cylinder cast being 3 cm. in length. The right bronchus was examined and there was considerable edema and some small ulcerations where the cast had been attached. The membrane had also involved the mouth of the right superior lobe bronchus and a few small shreds of membrane were removed from this region. The left bronchus was not involved. The usual

application of antitoxin swabbing was made to the involved area, and a long tracheobronchial tube introduced. The boy became very restless and delirious following the removal of the membrane, which had taken just seven minutes, and the temperature rose rapidly to 105 ; the pulse was imperceptible, and with extreme cyanosis and rapid respirations the outlook was very discouraging. The temperature continued to rise, which is often the case just before demise, and reached 105.6 at midnight. However, there was a sudden change, for the temperature began to fall during the early morning hours together with the respirations. The pulse was still weak and rapid. The next day, April 17, at 4 p. m., the long tube was removed. A curdy mass was coughed out following its removal. Three hours later dyspnea became urgent and Dr. Elerle intubated with a 4.5 O'Dwyer tube. This tube was auto-extubated fifteen minutes later and the patient was reintubated by Dr. Frick with a 6.7 tube. The tube was again coughed up one hour later and the same size tube was reintroduced by Dr. Frick. On the afternoon of the 18th, as the temperature was falling, the tube was removed by direct inspection to ascertain the cause for reintubation. There was supraglottic as well as subglottic edema, and the arytenoid cartilages were about three times their normal size. Because of these findings we decided not to interfere with the tube for at least one week and give the larynx a rest. This seemed impossible as the tube was repeatedly coughed up and ceased only after a 4.5 non-cough-up tube of the writer's model was introduced. The non-cough-up tube was not disturbed for five days and was then removed only after inspection of the arytenoid cartilages to see if the infiltration had subsided. On removal of the tube ventricular band and vocal cord spasm necessitated immediate reintroduction of the tube. The boy was at this time in fairly good condition and had recovered from the tracheobronchial diphtheritic attack but was unable to remain without the intubation tube. When the regular tube of Dr. O'Dwyer was introduced it was promptly coughed up, and the only tube that would remain in situ was the bulbous tracheal non-cough-up tube. On April 28, after having coughed up the regular O'Dwyer tube four times, a small swelling was noticed in the cricothyroid region of the neck and recognized as a beginning perilaryngeal abscess complicating perichondritis at the cricoid level. The neck was poulticed and when the abscess came to the surface it was incised and drained. The following day tracheoscopy was performed and ulcerations were seen at the cricoid level. No pus was draining into the larynx from the abscess cavity. On May 6, extubation was performed by Dr. Cannon, the Resident Physician, but immediate reintubation was necessary, the 4.5 non-cough-up tube being used.

It is an interesting fact that all of the cases of chronic laryngeal stenosis following diphtheria have similar histories of frequent auto-extubation during the first two or three weeks of the disease. On the other hand, however, we frequently meet with cases who wear the tube from four weeks to four months and subsequently are detubated permanently even when special laryngeal measures are not resorted to as an aid to detubation. During one year we had sixty-eight cases of the sub-acute type, as I classify them, and all of them made recoveries. Not one of this group became chronic. This, therefore, is the reason why we wait to see whether the case is to be chronic rather than perform tracheotomy, etc., when such procedure is not absolutely indicated as many of the cases of this type recover, as I have already stated.

without any special surgical measures. This case unfortunately became one of the chronics, and at the time this paper was read had worn tubes of special size and shape off and on for a period of one year. Repeated laryngeal galvanocautery knifing and puncture have been made to the subglottic region and intubation with soft rubber covered intubation tubes after cauterization. The boy has a remarkably good voice even after tubage of such duration and at the present time, April 15, 1917, seems to be apparently cured as he breathes well and has remained without the tube for a period of two months. We do not consider, however, any case as cured until the child has been detubated and is breathing well for at least a period of two years. Of course we are all hopeful that a cure has been affected but until the lapse of two years we shall not consider him cured. While the latter part of this history has no direct bearing on the title of the paper it is extremely important to report what does happen in some of those severe cases that recover and also to impress the fact that it is the inevitable nature of the pathological process at the outset of the disease which later is followed by the production of chronic cicatricial tissue.

*Case* 11. V. M., age two years, was admitted to the Willard Parker Hospital with a history of having aspirated a piece of egg shell three days prior to admission. The family physician who was called to see the child thought he was suffering from laryngeal diphtheria and notified the Health Department. As the child was dyspneic and required surgical care the parents were persuaded to send the child to the hospital. On admission to the hospital the family physician informed Dr. Gover of the history of the foreign body egg shell and further stated that the case was not one of diphtheria and he had changed his opinion after he had examined the larynx with a speculum and seen the foreign body. With this history at hand Dr. Gover examined the larynx with the infant speculum of Dr. Jackson and saw a small white substance at the level of the cricoid which he thought might be either egg shell or diphtheria membrane. As he did not wish to pass a bronchoscopic tube before I had seen the case, and as he knew that I was expected at the hospital, he very kindly left the task to me to decide the nature of the intruder. Through the direct speculum the same object was seen as noted by the family physician and Dr. Gover and both were eager for an opinion. The picture presented was not unlike many others and was thought to be diphtheritic membrane. The 4 mm. bronchoscope was carefully introduced and as it encountered the object it was very evident that it was not egg shell but diphtheria with which we had to deal. The small tracheal plaque was removed by suction and swabbing, a dose of 10,000 units of antitoxin administered and the child placed in the laryngeal ward. Intubation was not necessary, and the child made an uneventful recovery. Pharyngeal cultures were negative, but cultures made from the membrane showed Kleb-Loeffler bacilli.

A review of the cases demonstrates the fact which Dr. Chevalier Jackson has so often called attention to, that bronchoscopy and esophagoscopy can be performed without anesthesia, local or general, on adults as well as on children. Again, a patient without general anesthesia is much more of help than a hindrance to the operator and the operator loses no unnecessary time for there is no tendency to prolong the operation as it may be under general

anesthesia. Of course no case of diphtheria should ever be anesthetized for the removal of membrane. The profound carbonic acid poisoning from which all of these patients suffer makes a very good anesthetic for those who wish to satisfy their minds that general anesthesia is necessary. With the relief of this condition the child quickly returns to a normal state. Case 1 demonstrates the fact that an adult of 230 pounds can be easily bronchoscoped and the tube remain in situ for thirty minutes with practically no discomfort to the patient. In this case as in many of the others small doses of morphia and atropia were used. These drugs, more especially morphia, is the very best heart stimulant we have for this disease. Secondly, the atropia removes the greater part of the secretion which without it is often profuse. No doubt the accident in loosening the swab on removing the carrier has occurred many times before, but it does not happen since the long screw shoulder was added by Dr. Jackson. Case 2 demonstrates very forcibly the valve-like action of the loose membrane in causing over-distention of the lung with subsequent rupture of the alveoli and general subcutaneous emphysema, even when no instrumentation whatsoever has been attempted. It will also be noted that some of the moribund cases drowned in their own secretions. This was unfortunate even with continued suction. The profound carbonic acid poisoning and diphtheritic toxemia were also responsible. In two of the cases there was some difficulty in removing the membrane as the distal end was detached and flapping into the mouth of the tube while the proximal end of the cast was held tightly by the tube to the tracheal wall. This was in reality an overriding of the foreign body by the tube, which has so often been called to our attention that this error in technic should have been avoided.

The child with membrane causing obstruction below the distal end of the intubation tube will often when extubated breathe far better than when the tube is in situ. They will also regulate the amount of air taken with each respiration and breathe as quietly as possible. This is quite a common occurrence, and we have often recorded this shallow respiration as an endeavor on the part of the child to prevent coughing. On the other hand, when the membrane obstructs respiration there are violent spells of coughing accompanied by urgent dyspnea.

Case 7 demonstrated that direct extubation may cause supraglottic and arytenoid edema even when performed rapidly. This case was unable to remain without the tube when it was removed

through the speculum, and even when no infiltration was seen at the time of removal. At the suggestion of Dr. Thomas Joyce the tube was removed by the digital method. The patient remained permanently detubated after removal and had not even the slightest dyspnea or stenotic cough.

It is obvious that it is necessary to remove all of the obstructing foreign body membrane at the first attempt, but often this is impossible and the unfortunate child dies later of necrotic diphtheritic pneumonia.

In the tracheotomized cases in this series the same result followed as in all of my cases tracheotomized for the relief of tracheobronchial diphtheria; that is, they all terminated fatally. Of course all of these cases were moribund with the exception of case 9, who lived for thirteen days. All of the membrane was not removed from this child at the first attempt and the fatal result was due to necrotic pneumonia.

We have had a number of cases with foreign bodies admitted for diphtheria and treated for this disease in the hospital and the cause was discovered only after Dr. Gover or myself had made a direct examination of the larynx and trachea. The exception to the rule is case 11, who was admitted with a definite history of a foreign body and by direct inspection was definitely proven to be suffering from tracheal diphtheria.

## DISCUSSION.

**Dr. Samuel Iglauer**, Cincinnati, Ohio, was interested to note that Dr. Johnston did not hesitate to use atropin or morphin when he thought them to be indicated. If the secretion could be dried up by these measures they should be employed. He was also interested to know that Dr. Johnston had reached the point of being able to employ direct laryngoscopy in the home. This would be a great aid. He recalled one case in which he suspected laryngeal diphtheria. In order to make the examination he had to take the child to the hospital. Both Dr. Johnston and Dr. Jackson advocated the use of small tubes; he had always thought he could see more through a large tube. It was sometimes necessary to devise instruments to meet the needs of special cases.

Referring to Dr. Lynah's remarks concerning emphysema of the lung in bronchial obstruction, the speaker recalled that some years ago he had pointed out that with a partially obstructive foreign body in the bronchus the lung of the same side might become over-distended, owing to the fact that air passed the foreign body during inspiration but was trapped when the bronchus contracted during expiration. The resulting distention of the lung on the foreign body side might lead to error in diagnosis as one usually expected lung collapse, and not distention beyond a foreign body.

**Dr. Jackson**, in closing the discussion, emphasized the importance of Dr. Johnston's work, which, in the speaker's hands, had proved to be all that Dr. Johnston had claimed for it. Direct laryngoscopy would never come into its full degree of usefulness until the laryngoscopist

was prepared, as Dr. Johnston was, to go into the home and tell the general practitioner why the child had a croupy cough. If diphtheria was suspected, the family physician would not allow the laryngologist to take the child to the hospital and give it an anesthetic.

Referring to Dr. Lynah's remarks about the use of small tubes, the speaker deprecated the use of large tubes. Unfortunately much preliminary practice had been done in clinics on adults under local anesthesia. Then when the operator encountered a foreign body case in an infant he was handicapped by being compelled to work through a very small tube. If the use of a large tube were attempted it would prove fatal to the child, as shown by the work of Dr. Imperatori and Dr. Forbes.

There was little objection to using the drugs referred to by Dr. Iglauer in adults or adolescents. In children, and especially in infants, morphin and cocain were both very dangerous.

As to bronchoscopy in cases of abscess in cases other than those of foreign bodies he had seldom been able to reach anything that could be called an abscess cavity. Usually the pus was found to come from a small branch bronchus, the orifice of which was swollen and more or less inflammatory. A further study was being made of such cases.

# THE PRESENTATION OF A CASE OPERATED FOR LARGE SUBDURAL CYST OF THE FRONTAL LOBE.

By G. W. MACKENZIE, M. D., Philadelphia, Pa.

My object in presenting this case is to show to what extent a careful eye, ear, nose and throat examination aids one in the diagnosis of intracranial conditions. Practically all of the information obtainable as to the patient's condition was through these sources, as a study of the report will show.

*Patient:* Wm. S., age 57. Was referred to me by Dr. H. M. Goddard on March 5th, 1917.

*Family history:* Mother died at the age of 87. So far as patient can recall, her general health was good for a number of years before her death. Father died also at the age of 87. His general health had been good with the exception of an attack of rheumatic fever when 50 years of age. Patient is one of a family of 13, most of whom lived to a ripe old age; one brother died from an acute cardiac condition; cannot recall the exact cause of death of any of the other brothers. He has a sister still living, past 60 years of age.

*Previous history:* Patient cannot recall any definite diseases excepting those of childhood, which were few. At the age of 21 he had, what the doctors termed, inflammation of the bowels and catarrh of the stomach for a period of six or seven weeks (probably typhoid). At that time he was employed as a carpet weaver. At the age of 26 he married, and is the father of eleven children. At the present time there are six children living. One child died from diphtheria, another from pneumonia, both before they were ten years of age. Cannot recall the cause of death in the other two. The first child was still-born.

*Present history:* In the early part of October, 1916, the patient fell downstairs, was picked up unconscious and moved to bed. The following day the family doctor was called in. At that time the patient complained of headache over the right occipital region. A day or two later he returned to work and felt all right for about one month, when the headache returned. Since then the pain had become more intense, but the intensity varied from time to time. Patient consulted an osteopath and received nine treatments. He then called in a general physician, who consulted with another physician, who was more or less of a specialist. He was then transferred to one of the Philadelphia hospitals and was under the care of a surgeon for about three weeks. The patient appeared to be benefited by the treatment and left the hospital of his own accord and returned home. He remained under the care of his family physician, who later sent him to another hospital where he was under treatment for about nine days. During this time a polyp was removed from the right side of the nose and a frontal sinus condition diagnosed, which was thought to be the probable cause of the patient's headaches. During his stay at the second hospital the patient claims to have become gradually weaker as the

result of a restricted diet. He then consulted Dr. Goddard, who referred him to me on March 5th, 1917.

*Present condition*: The general appearance of the patient was that of pronounced emaciation; he had a shuffling, uncertain gait (length of steps about eight inches), requiring two persons to lead and support him. His speech was slow and he had a vacant stare in his eyes, which suggested poor vision. He complained of severe pain on the right side of the head, centering especially in the temporal and frontal regions. During his attempts at walking, there was a tendency to pitch forward due to an inability to balance himself. The same tendency was manifested if one pushed his body backward or to either side.

Examination of the cranial nerves gave the following findings:

I. Nerve: The patient was unable to distinguish between the odor of alcohol and catsup. His answers on repeated tests, with a few minutes' rest between, were occasionally given correct, but were always slow. Each side was tested separately and the patient's sense of smell seemed to be blunted on both sides. On the left side the blunting of the sense of smell may have been due in part to a pronounced obstruction from a deviation; the right side (concave side) was quite free of mechanical obstructions, however there was considerable secretion. At the time of taking the sense of smell there was some question in my mind as to how much was due to an actual blunting of the sense of smell, how much to obstruction and how much to slow cerebration. Later on, I learned that the patient, under normal conditions, prior to and since operation, is a slow and deliberate talker, but he is more prompt in his replies at this time (June, 1917) than he had been for some time prior to operation.

II. Nerve: Ophthalmoscopic examination O. D. Optic nerve showed extreme choking (elevation 6 or 7 diopters), as well as pronounced increase in circular dimensions (mushroomed). The outline of the disc was very poorly defined, apparently blending with the retina. The vessels on the disc were quite obscured, the veins in the retina markedly distended. The retina showed innumerable streak-like small hemorrhages, mostly surrounding the veins, while at the lower part of the retina there were some larger masses of hemorrhage; the upper margin of these were horizontal. There were a few small areas of pale-colored degeneration scattered between the disc and the macula. The distribution of these areas, however, was not of the classical albuminuric type. The vision O. D. was fingers against a black background about one foot (?), more often incorrectly estimated than correctly. O. S.—the ophthalmoscopic picture was the same as that of O. D. with the exception that the choking was less pronounced (4 or 5 diopters), the hemorrhages perhaps fewer, while the vision was about the same as that of O. D.

III., IV. and VI. Nerves: Mobility of the eyeballs normal, in that both eyes moved together and freely in all directions, with no evidence of strabismus; however, on examination for the lateral positions, there was an apparent aggravation of the physiologic nystagmus. Pupils were both moderately dilated, the right more so than the left. Reaction to light was practically nil on the right side, but moderate on the left.

V. Nerve: No disturbance in tactile, pain or temperature sense on the two sides, nor was there any weakness of the muscles supplied by the motor branch of the V. nerve on either side.

VII. Nerve: The function of the mimic muscles on both sides equal and normal.

VIII. Nerve, Cochlear Branch: Hearing for conversational and whispered voice and acoumeter apparently normal on both sides. *The fork tests gave normal findings in that Weber was not lateralized. The bone conduction was normal, Rinné positive 40 seconds, which is normal for the Politzer middle tone weighted fork that I use. Patient also heard the low and high forks normally.

VIII. Nerve, Vestibular Branch: The extreme headache which the patient was suffering, together with his inability to sit in the turning chair, made it impossible to conduct turning tests. Accordingly, the galvanic test, which I believe to be an easier and more accurate method, was used. With the cathode to the right ear, the patient manifested a rotatory nystagmus to the right with a current strength of 3 M. A. With the anode to the right ear, there was a pronounced rotatory nystagmus to the left with a current strength of 3 M. A. On the left side the cathode produced a nystagmus to the left with 3 M. A. current; while the anode produced a rotary nystagmus to the right with 3 M. A. Prior to making these galvanic tests, the patient was tested for spontaneous nystagmus, which was not present when looking straight ahead. On looking to the right, there was pronounced nystagmus to the right, and on looking to the left, there was pronounced nystagmus to the left, as noted above; however, it was equal in intensity to the two sides. The disturbance in gait has already been mentioned.

There was spontaneous past-pointing inward, about 8 inches on both sides; that is, when the patient attempted to find the finger from above downward, he deviated inward with the right hand; also with the left hand, and the condition was to a like degree on both sides.

IX. Nerve: The sense of taste was tested with salt, sugar, alcohol and catsup, all in liquid form, with a small pledget of cotton wrapped on an applicator wire. The two sides were tested separately and the tips and the dorsum of the tongue were also tested separately and the mouth washed with water between tests. The replies were very uncertain and tardy. Comparing them with the control, they were much slower than normal, especially on the left side; however, there was not a pronounced variation on the two sides.

X. Nerve: Temperature 98°, pulse 64, respiration 24, and no vomiting up to the time of date of this examination; however, there was one attack of what the nurse reported as expulsive vomiting as the patient reached the hospital and prior to his operation.

XI. and XII. Nerves: Normal findings.

The general muscular condition of the patient was fair and equal on the two sides; that is, the extensors, flexors, supinators and pronators were tested and found to be weaker than the average but equal on the two sides. The same applies to the hand grip.

The arms, legs, chest and back were tested for disturbances in the tactile, pain and temperature sense, but none were found.

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*In regard to the tuning-fork tests, they were rather difficult to make, because the patient seemed to fatigue under the tests and it was necessary to complete the examination at the second attempt two days later. At times there was a question as to whether there was not slight diminution on both sides of very moderate degree, but repeated tests, using considerable patience, showed that the function of the cochlear branch of the VIII Nerve on both sides was normal.

The speech centre was found to be normal, in that there was no motor or sensory aphasia nor was there any paraphasia; furthermore, the family was questioned on these points and replied that they had not noticed any disturbance in his speech or in his identification of articles. On the other hand, there was a slight hesitancy in his speech that the family had noticed which the patient did not show before his present illness.

Examination of the nose revealed a pronounced deviation of the septum to the left, as noted. On the right concave side there was a moderate compensatory hypertrophy of the inferior turbinate and a moderate amount of secretion in the region of the middle meatus. The appearance of the stump of the middle turbinate suggested that it evidently had been amputated, because of previous hyperplasia. The mucous membrane on both sides of the nose was somewhat pinker and more turgescent than normal.

Examination of the mouth revealed three scattered wounds in the alveolar process of the upper jaw, due to the recent extraction of teeth. The tongue was slightly coated, of a gray color, and was protruded straight forward with a slight, coarse tremor. He could extend his tongue and move it to the two sides freely and to a like degree. The soft palate moved freely and did not deviate during the phonation of the letter a.

*Otoscopic examination*: Right ear—tympanic membrane intact, brilliant, fairly opaque, no retraction, normal mobility with Siegle. By Politzer inflation, the membrane moved outward freely over a large area and went back somewhat slowly, especially in the posterior half, indicating a somewhat relaxed condition of the membrane.

Left ear—Membrane intact, brilliant, slightly opaque in the posterior half; however, sufficiently translucent to permit a view of the long process of the anvil. Mobility normal with the Siegle. By Politzer inflation the membrane moved outward freely and went back promptly.

*Urinary examination*, March 5th: Amount, 4 ounces. Sp. Gr., 1009. Reaction, faintly acid. Odor, none. Color, light amber. Transparency, clear. Sediment, heavy grayish white flocculent. Albumen, trace. Sugar, negative. Urea, 0.010 gms. per c.c. Indican, negative. Bile, negative. Urobilinogen, negative. Blood, negative.

Microscopic—An abundance of amorphous urates, many epithelial cells (mainly bladder), few renal cells, an abundance of bacillary bacteria, few uric acid crystals.

*Wassermann examination*, March 13, 1917: Made by the Philadelphia Clinical Laboratory. Reaction, negative. Antigen I., (Cholesternized) negative. Antigen II., (Alc. syph. liver) negative. Antigen III., (Acet. insol. lipoids) negative.

From the symptoms and signs the diagnosis was made of a tumor of fair size, located in and about the right inferior frontal convolution.

The patient was sent to the hospital for further study before attempting an operation. I visited him several times, corroborating the findings noted above. On the 15th of March I took Dr. Weston D. Bayley, of Philadelphia, as consulting neurologist, his findings corroborating those of mine. Dr. Bayley furthermore agreed with me as to the character and location of the lesion. On March 16th the patient was operated under ether narcosis, administered by Dr. R. Frank Hill. A semicircular incision was made on the right side extending from a point an inch behind and on a level with the auricle, upward and around

to the outer angle of the orbit, through the soft parts and periosteum to the bone. The periosteum was separated from the bone and the flap turned downward. With the Alexander mastoid gauge No. 12 an opening was made through the skull in about the middle of the exposed area. This was enlarged with the bone-biting forceps in all directions and an oval-shaped area of the dura exposed about 7 c.m. in its longest dimension, (horizontal) and 6 c.m. in its shortest dimension (vertical). The dura appeared normal and smooth on its external surface, but beneath could be seen a dark discoloration. Furthermore, there was evidence of marked pressure. A small horizontally running incision was made anteriorily through the dura, from which squirted a dark, almost black-colored, fluid. This was allowed to flow slowly until it appeared that a sac had collapsed. Then the incision was lengthened as far as the exposed dura would permit. The upper margin of the dura was retracted and it was seen that there was a considerable space between the dura and the brain tissue, giving one the impression that the brain had suffered a dent, the maximum depth from the surface of the dura to the brain tissue being about 2 c.m. About a dessert-spoon full of dark clots was removed, after which an inspection of the dura and surface of the brain showed a dark greenish-colored stain,—no doubt, from the iron salts of the blood. The dura over the cyst was about three times as thick as normal, the inner two-thirds of which appeared to be a more or less organized sac which could be peeled from the dura proper with some difficulty. The greater part of the dura was stitched, with the exception of a small gap which was left anteriorly, into which the end of a one inch iodoform gauze strip was inserted for drainage. The wound over the soft tissues was closed with interrupted sutures, except the extreme anterior end, through which was allowed to protrude the iodoform gauze wick. Iodoform gauze dressing was applied directly to the wound, over which a rich plain gauze dressing was laid and the whole bandaged. The patient was put to bed and a few hours later, when he had come out of ether, he remarked that his headache had gone.

On March 17 the patient's pulse, temperature and respiration were normal. There was slight edema about the right orbit; otherwise, he said that he felt comfortable and remarked that he could see better.

On March 18th the patient's condition was improved from every angle. The wound was redressed and showed no evidence of excessive reaction. After this date the wound was dressed every day. The headache had entirely disappeared and he noticed that his vision was constantly improving.

On March 19th, the wick was partly withdrawn and the following day removed entirely. At this time his gait had so improved and he felt strong enough to walk from the ward to the operating room for redressing. On the 24th the wound was quite healed. There was no secretion, his vision had improved and he was discharged from the hospital. Two weeks after the operation, the patient walked from his house to the office, accompanied, but unassisted, a distance of about three miles.

On March 28th the patient was re-examined, and the sense of smell, although not perfect, was much better than on his first visit. The optic nerve, O. D., was still swollen about 3 diopters. The blood vessels on the disc were seen more distinctly than they were on the first visit. Hemorrhages were considerably diminished in number, and the patient's vision improved to fingers at one meter. O. S., disc swollen about 2 diopters, the same general appearance as

in O. D., but less pronounced. Vision, with correction that he had been wearing 6/30.

The sense of taste was positively improved over that which it had been prior to operation.

The remaining cranial nerves normal. The patient had gained 10 pounds in weight and was physically much stronger than before operation, and entirely free of headache.

On April 21st K. I. was administered in 10 drop doses, t. i. d. in the hope of clearing up the slight remains of edema of the disc. The patient reported at the office about twice a week from then on until May 19th, when he was refracted with a mydriatic. The vision in right eye was 6/60. It was impossible to improve the vision in this eye with any glass. O. S. + 1.87 D sphere + .50 D cyl. ax. 105 gave a vision of 6/12. With 4.00 D sphere added, the patient was able to read ordinary newspaper type. The patient observed his central vision was poor, as compared with that directly around it. He stated that he loses fixation of objects when he looks directly at them, but when he directs his vision away from the center, he reads better than when looking directly at it.

The urinary examination made on May 5th was as follows:

*Urinary examination,* May 5, 1917:..Report of the Philadelphia Clinical Laboratory—Color, light yellow. Consistence, clear. Reaction, acid. Sp. gr. at 15° C.—1018. Robert's test for albumen, not found. Robert's test for heat + H No. 3 not found. Benedict's test for sugar, not found. Casts, not found. Urates amorphous, present. Crystals, not found. Large squamous epithelial cells, few found. Leucocytes, few found.

By ophthalmoscopic examination there was no evidence of swelling of the disc, distention of the veins or hemorrhages.

Since May 19th the patient reported every week or 10 days up to June 16th. His vision is still improving, so that now he reads 6/12 with ease, and is discharged as cured.

The points worth emphasizing in this case are: 1st, that the very severe headache combined with the immense choking of both optic nerves, were sufficient to indicate that there was considerable intracranial pressure; 2nd, that the headache was more intense on the right side; 3rd, that the right optic nerve was choked more than the left: 4th, that the vision was poorer in that eye;—all pointed to a right-sided lesion.

The absence of localizing symptoms with the exception of the sense of taste and smell, would suggest that the location of the pressure was in some so-called silent area in the region of the inferior frontal convolution of the right side, just above the Sylvian fissure anterior extremity, and possibly exerting some pressure on the anterior portion of the temporo-sphenoidal lobe.

That there was no speech disturbance was due to the fact that the patient was right-handed.

The presence of the spontaneous past pointing inward with the right and left hand, together with the exaggerated physio-

logic nystagmus to the sides, were probably due to distant pressure and were of no particular localizing value.

The history of a fall in November, from which time the patient dated his trouble, would suggest that the character of the lesion was probably hemorrhagic.

# INTUBATIONAL TUBES FOR THE TREATMENT OF CHRONIC LARYNGEAL STENOSIS.

By HENRY LOWNDES LYNAH, M. D., New York City.

A. *The Dilating Laryngeal Tubes.*

These tubes are made in five sizes, with an increase of one millimeter in the antero-posterior, as well as the lateral diameter, over the original O'Dwyer tube. The first size is introduced after the O'Dwyer tube is removed. The tube is removed at the end of one week and the next size introduced. For greater dilatation these tubes are covered with soft rubber tubing, which gives an increase of from one to two millimeters in diameter. The tube in the middle of the row of dilating tubes shows one covered with the soft vulcanate.

B. *The Non Cough-up Tubes.*

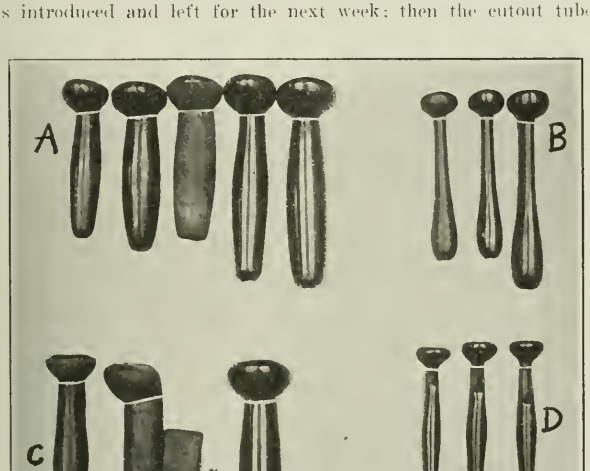
These tubes are used in acute laryngeal diphtheria to prevent auto-extubation, and are also employed in the chronic cases of stenosis when auto-extubation occurs. They are also used owing to the very narrow neck—to allow for play of the intralaryngeal muscles after sufficient dilatation has been accomplished. They prevent auto-extubation owing to the olive bulbous tracheal swell In acute laryngeal stenosis they should not be left in longer than one week, as there may be considerable difficulty in removing them. These tubes have been used in over three hundred and fifty cases of persistent auto-extubation, and in not one instance has the tube been coughed out after introduction. These tubes are made in seven sizes—from one to ten to twelve years of age.

C. *Intubational Laryngostomy Tubes.*

These tubes are employed when the radical laryngostomy is necessary to effect a cure. The laryngostomy is performed in the usual manner, care being taken not to disturb the dermal flap on introduction of the tube. These tubes are covered with soft rubber. After introduction of the tube a large soft rubber laryngostomy plug is attached by means of a small threaded post with a pressure flange attachment, so that the soft rubber plug may be pressed down and made to fit the fistula and keep the lateral walls separated.

D. *The Abductor or Cutout Tubes.*

These tubes are made with a very flat head and the posterior portion of the lumen removed. The removal of this part of the tube allows free play for the abductor muscles after a small narrow neck tube has been used without success. The first size is introduced and left in situ for one week, provided it does not cause dyspnea; then it is removed and a narrow neck tube, without the fenestra, is introduced and left for the next week; then the cutout tube




introduced and left for another week. At the end of the second week the next size fenestrated tube is introduced alternating with the non fenestrated narrow neck tube, and so on with the next size until a period of six weeks has elapsed, when the tube is removed for detubatory trial. If the child does not remain without the tube and spasm of the adductors is still the cause of reintubation, the same procedure is repeated. If polypoid tissue is present these tubes should be dispensed with, or rather not employed. All of

the cases should be constantly examined by endolaryngeal inspection to ascertain the cause of the stenosis before blind digital intubation is attempted.

These tubes are made by George Ermold of New York City.

## PRESENTATION OF SPECIMENS.

Dr. Robert Clyde Lynch, New Orleans, La., presented a specimen, the larynx, removed from a male patient, 72 years of age. The man was first seen in May, 1916, when the diagnosis of carcinoma of the larynx was made, and operation advised. This was refused, and the patient passed out of his hands. Radium was then used: In May, 77 milligrams, for seven hours; in June, 80 milligrams, for 10 hours; in August, 77 milligrams, for fourteen hours.

The first few applications of radium produced ulceration of the skin surface; this healed. The tumor increased in size, obstructing the air-way. He then returned, begging for relief. Laryngectomy was thereupon performed, the one-stage operation being employed. The operation progressed satisfactorily in all respects, but the tissues over which the radium had been applied were found to look exactly like cartilage, being hard, gristly, and entirely different from the normal condition in the neck. Over the area of application and beginning in the tumor itself, there was some necrosis from the action of the radium. Immediately after the operation there was set up radium sloughing, which led to the patient's death.

This case was presented because there had been several similar cases in New Orleans, in which, because of too large doses or insufficient screening, operation was followed by tremendous sloughing. In one case, in which radium was applied to the thyroid, the woman had sufficient strength to bring about her recovery. The cases illustrated the fact that where radium applications preceded the operative procedure, one must be prepared to deal with sloughing of the tissues. The action of the radium on the normal structures was to be taken into consideration.

MEETING OF THE EASTERN SECTION, HELD IN PHILADELPHIA, PENNSYLVANIA, MARCH 3rd, 1917, UNDER THE CHAIRMANSHIP OF DR. JOHN F. CULP, OF HARRISBURG, PA.

## CHONDROMA OF THE SOFT PALATE LONG OVERLOOKED.

By BEN CLARK GILE, M. D., Philadelphia, Pa.

It sounds like a paradox to say that we need to be reminded of familiar truths, but this is a fact. Every physician knows that a correct diagnosis is invaluable and that we should earnestly strive to discover and remove the causes of the diseases we encounter and yet quite frequently we learn of cases, whose treatment has been exclusively symptomatic and where etiological investigation has been entirely neglected. Of course, we are often required to treat symptoms: it may be our first duty to check a hemorrhage or relieve a severe pain, irrespective of causation but, when we have time for investigation, especially in chronic cases, purely symptomatic treatment is justified only by some one of the following conditions: (1) where the cause cannot be discovered; (2) where the cause is a self limiting process, as in many of the disorders occurring during pregnancy, and (3) where the cause though known, is beyond remedy and must be reckoned with as a permanent factor. Apart from situations such as these, the restriction of our therapy to the palliation of symptoms is negligence hard to condone.

In the case I am about to describe, a slowly growing tumor, in an accessible locality, had reached a considerable size and had given rise to a number of symptoms. The patient had been successively under the care of four practitioners, two of them laryngologists, and various remedial measures, medical and surgical, had been employed, but all treatment had been addressed to symptoms: no one did anything with the causal tumor and, in truth, only one of the four scrutinized the region where it was to be found. The varied treatments temporarily palliated the symptoms, but had no permanent effects and the patient was growing worse until, at last, the tumor was recognized as the sole cause of the morbid conditions, and was extirpated, bringing about complete recovery.

As some of the statements in this narrative may seem strange, I wish to be particularly clear and definite regarding the authority upon which they rest. Nothing which transpired before I took charge of the case came within my own personal knowledge.

When the patient consulted me, she had already parted company with her former advisers and I had no opportunity to confer with them. The patient is a young woman of intelligence and I believe her to be entirely trustworthy. On account of her prolonged treatment and large outlay of money, this illness was for her a very trying episode and she put in writing a history of her experiences, from the time when she first sought medical aid until its final cessation, upon her recovery of health. It is upon this record I have depended for the facts preceding the time when I made her acquaintance. I believe that, to the best of her ability, she has narrated with accuracy what occurred.

## CASE REPORT.

In the year 1911, Miss E. W. about 30 years of age, sought the advice of her family physician, because of persistent earache upon the right side. There was no discharge and hearing was not impaired. The throat was not examined and the treatment consisted of internal medication. The pain gradually passed away.

In 1913 there was another prolonged attack of earache and an intercurrent tonsillitis. Both subsided in a couple of weeks but left a cough, which continued for months. The same physician was in charge and the patient took many bottles of medicine; character unknown. There were no surgical procedures.

In 1914 a very painful abscess developed in the right ear and was lanced by Dr. X. a laryngologist. No examination of throat or nose. Later in the same year occurred another attack of tonsillitis with a troublesome cough. These affections were treated by Dr. Y. who administered a good deal of medicine by the stomach. No surgery and no examination of nose or of naso-pharynx.

Near the end of 1914 the patient, much discouraged by her frequent attacks of illness, applied to a laryngologist, Dr. Z. and was under his care for eight months, extending beyond the summer of 1915. There was no surgery but medicines were applied to the throat and nostrils. With warm weather the symptoms were somewhat mitigated.

In April, 1916, there was another abscess in the right ear accompanied by a persistent cough. The patient returned to the laryngologist, Dr. Z., who lanced the abscess and also inspected the soft palate, as far as that could be done by simply looking into the mouth without any instrumental aid, or any attempt to take a posterior view. Inadequate as this examination was, it showed that the soft palate was distended by tumefaction of some kind and Dr. Z. informed the patient of the abnormal condition which existed, adding that he did not know what the growth was and did not think it wise to interfere with it. A Wassermann test was made with a negative result; but the Eustachian catheter was not used nor were the Valsalva or Politzer methods of inflation. The patient continued to visit Dr. Z. up to August, 1916, when swelling again appeared in the right ear and lancing brought temporary relief; but the trouble soon returned and Miss W., in a discouraged

frame of mind, was looking forward to another repetition of the incision, when the condition of a tooth led her to consult Dr. Briggs, a dentist in whom she had confidence. This was a fortunate incident, for Dr. Briggs assured her that the throat was seriously affected and required radical treatment to reach the cause of the recurrent disorder in the ear. At his solicitation she came to my office upon September 30, 1916.

At that time in addition to mental and physical depression, the patient was suffering from a rasping cough and her throat was sore, though not actually painful. Her chief discomfort was in the right ear, where there was a sensation of tension and stuffiness and she was strongly impressed with the notion that the time had come for another cutting of this much-incised organ. Tests of the hearing showed that it was decidedly impaired upon the right side and they also localized the trouble in the middle ear. Examination with the aural speculum indicated that there was considerable liquid in the right tympanic cavity, escape being prevented by occlusion of the Eustachian tube on that side.

The anterior view of the fauces showed, at the first glance that the soft palate was in a very abnormal condition. It was distorted by some internal pressure causing it to bulge forward. The quinsy points were conspicuous and, showing through the mucous membrane, were small nodules pale in color, firm and hard, but not tender under the touch. The postnasal mirror showed a small chink behind the soft palate on the left side; but the right was filled up by a swelling projecting from the soft palate into the nasopharynx and preventing the finger from entering the postnasal space. This adventitious structure was firm and gave no sign of inflammation. Neither of the Eustachian eminences could be seen; inflation by the Politzer method succeeded on the left side, but failed on the right. The eustachian catheter passed readily along the nasal floor but, when its beak reached the soft palate, it met an obstruction and could not be advanced further nor could it be rotated.

These examinations proved that a tumor of considerable size was imbedded in the soft palate and from the signs given to sight and touch, combined with clinical history, I came to the conclusion that it was a chondroma, a judgment which was corroborated by the laboratory tests made subsequently. I informed the patient of what had been found, telling her frankly and firmly that the best treatment, indeed the only proper treatment, was the entire removal of the tumor: that this course would give her good prospect of recovery; but that partial, half-way measures would be of no advantage. She took a sensible view of the matter and agreed to a prompt operation.

Just here let me say that I claim no credit for recognizing this neoplasm. It was in an accessible locality and could be distinctly seen by pharyngoscopy. Any physician should have made himself familiar with its chief features upon his first inspection of the throat. The puzzling questions are why one specialist never looked for it and why another, after seeing it, told the patient that he did not know what it was and considered it best to let it alone.

Two days later, October 2, 1916, the operation was performed under general anesthesia induced by ether. The right tonsil, which was somewhat morbid, was enucleated and the wound enlarged until it reached the level of the upper margin of the chondroma. This was then dissected out. When its attachments had been severed, it was seen to have a nodular structure and came away in three pieces. Taken together these three weighed a little more than

an ounce. Bleeding was controlled by pressure and the post operative condition was normal.

The patient had a rapid convalescence. Although the right ear received no topical treatment, hearing became absolutely normal in 48 hours. The cough showed prompt improvement and in ten days ceased entirely. Healing was complete upon the sixteenth day; there remained no disability of any description.

Five months have now elapsed since the operation and no morbid sequels have appeared. I saw the patient within a week and she was in excellent health and spirits. There can be no doubt that the chondroma was the cause of all the varied symptoms from which she suffered during six years and whose persistence threatened to bring her to invalidism.

I have given the history of this case thinking that it may prove both a caution and an incentive. We are all liable to err and if oversights, such as those narrated are frankly criticised, this is not done with a purpose to humiliate our colleagues, who appear to have been at fault; but in the hope that all of us may be stimulated to renewed appreciation of the importance of thoroughness in search for causation.

Only by making a correct diagnosis and directing our treatment to the radical removal of the cause of disease can we do our full duty to our patients and attain that high professional ideal, which combines wide knowledge of medical science with expert proficiency in the honored specialty, which is our chosen vocation.

## DISCUSSION.

**Dr. Coffin:** The doctor states that these cases are not uncommon, but I rather feel that they are. Personally I have seen this condition once. I think men are fairly excusable for not making a diagnosis at first. In not making a diagnosis at all they are not excusable. The case that I saw had been brought to me for consultation by a man who had been working in laryngology almost as long as I had. He had this growth exactly as Dr. Gile describes it. I had not heard Dr. Kyle's paper. I could not make a diagnosis at the time, but I did before the patient got away. I practically went at it as though I was to remove a tonsil, and came across a growth into which I could poke a separator. I scooped it out until I got soft tissue all about.

**Dr. Gile.** In reply to question: I made my diagnosis with the aid of my memory of a paper and specimens that I saw presented by Dr. Kyle at the College of Physicians. The tumor in my case was hard and nodular, but not painful. As I had gone carefully over the diagnosis I concluded this was a chondroma.

(Replying to Dr. Worthington): When the patient opened the mouth the tumor was exactly where a quinsy points. When I operated I put my finger in the post-nasal space and came to the conclusion, with the aid of the mirror, that the chondroma had split the soft palate and forced the nasopharyngeal wall of the soft palate up against the eustachian tube. It had a mucous membrane surface anteriorly and posteriorly.

(Replying to Dr. Cohen): The firmest attachment of the tumor seemed to be the submaxillary angle of the jaw. After the operation I was not able to satisfy myself exactly on this point. It seemed that the region of the sphenoid bone was flattened. It did not spring from the eustachian tube.

## MILIARY TUBERCULOSIS SIMULATING FIRST, FRONTAL SINUS INFLAMMATION AND SECONDLY, A BRAIN LESION.

By GEORGE L. RICHARDS, M. D., Fall River, Mass.

The following case is of interest as showing how misleading the apparent symptomatology may be and how necessary a careful physical examination is in all doubtful cases and even in many of those which on first examination seem to be reasonably clear as to diagnosis.

Patient J. M., age 25, Portuguese, weaver. Came into my office late on the evening of Thursday, December 21st, 1916, complaining of a severe headache over both eyes but very much worse over the right, of two weeks duration.

He had previously consulted a reputable general physician who had been unable to locate his trouble, and thinking it might be an involvement of the frontal sinus, referred him to me.

The patient spoke English very poorly, as did also the friend who accompanied him, hence, about the only definite knowledge we could gather was pain in the region of the frontal sinus and supraorbital area.

Transillumination showed all the sinuses clear. Fundus examination showed a very slight papillitis on the right side, nothing on the left, no nasal discharge. Blood pressure 130. Temperature 100½ . Urine a slight amount of albumen, a few blood cells but no casts.

He was given local treatment, a cathartic and aspirin, sent home and advised to return Saturday afternoon and was requested to come early in the day and bring an interpreter with him.

Saturday he returned, but again very late in the afternoon because the interpreter could not leave his own work earlier. The patient now looked very ill indeed, in fact too ill to pay attention to any questions put to him. The interpreter stated that he had been growing rapidly worse until the pain in his head was very excruciating. Nothing he had been able to do or take had in any way relieved his symptoms. We were told that the man had lived in this country two years, and had never been ill up to two weeks previous, when the present attack came on. The only

symptoms then or at any time since had been frontal headaches. (Just how much reliance could be placed upon this history is doubtful, as at no time did the man seem to realize or understand much of what was being asked him by the interpreter, and his cerebration seemed slow from the very first).

A cab was called and he was sent to the Union Hospital. The next day, Sunday, he was worse, still complaining of "pisa." Portuguese word for pain, and pointing to his forehead. Transilluminations were still negative as were also the X-ray plates. He was really too restless to allow of a good X-ray plate. All symptoms seemed to indicate frontal sinus pressure, and in the hope that with the release of air pressure or perhaps pus, the patient might receive some comfort, he was taken to the operating room Sunday evening and under local anesthesia the right frontal was opened intranasally. The results were negative except for a few hours lessened pain due probably to the cocain.

The next morning, Monday, the pain had returned with its usual intensity and the man began to become markedly comatose. Upon entering the hospital the blood showed a leucocyte count of 8,000, differential count was normal, the third day the white count was 16,000, and on the fifth day, again 8,000. Wassermann was negative. Widal negative. Noguchi positive. Spinal fluid negative on pressure and a microscopical examination and a culture of this were also negative. The Wassermann was done on the blood and the Noguchi on the spinal fluid.

The radial pulse was slow at all times, the cardiac pulse being about twice that of the wrist. He was very restless and got out of bed and passed his urine on the floor.

The medical men of the hospital as well as the neurologist went over the patient thoroughly and repeatedly, all agreeing that the trouble must be some form of intracranial pressure. In view of many of the symptoms to wit: insomnia, severe localized headaches, increasing at the close of the day, slow pulse, decidedly slow, difficult cerebration with periods of intelligence, very sick expression, slight papillitis, involuntary urination, etc., the above diagnosis could not be entirely ruled out, but in consideration of the negative findings such as negative ear findings, no history of ever having had any ear trouble, at no time vomiting or nausea, slight fever, moderate pulse, irritability only when actually touched, no paresis of either upper or lower extremities, asked to squeeze the hand, he would hesitate for a minute or two and then perform the act with considerable

strength, no muscle spasm, no motor oculi paralysis, no ptosis, no pupillary changes, in fact no local motor or sensory paralysis; a definite cerebral diagnosis seemed impossible. I did not, therefore, feel justified in making an exploratory intracranial operation although permission for such had been obtained. I did not, however, feel comfortable at having done only an intranasal opening of the frontal sinus as the pain at all times was in this region, so he was again taken to the operating room and under general anesthesia his frontal sinus was opened externally. A perfectly normal sinus was found, no exudate, no inflammation, no polyps. There seemed to be nothing else to do but to await results. Patient died the following morning.

Autopsy showed the following:—

Brain, no adhesions of the dura to the bone, no depression, no general blood extravasation, no exostosis or fractures, no acute inflammatory lesions. I say acute because over the right parietal lobe there was an area about the size of a dollar which showed an exudate resembling pus but on the incision of the membranes through this area, it was found not to be pus but a cheesy tubercular like deposit. On making an incision through the brain substance half an inch below this area there was found a small spot about the size of a silver quarter which looked microscopically very much like a small hemorrhage. Sections of this area did not prove a hemorrhage but rather a chronic process marked by round cell infiltration almost entirely free from polymorphonuclears. This was absolutely the only abnormal finding in the brain except that the entire brain substance seemed slightly softer than usual.

Chest, Lungs. Left pleura was markedly adherent to the chest wall. The lung substance showed numerous miliary tubercles small and irregular in shape and distributed throughout the entire lung. There was scarcely an area the size of a finger tip that did not show these tubercles. Right lung, pleura not adherent but the lung substance showed many tubercles especially in the upper and lower portions. Sections of the lung showed multiple tubercles which had largely replaced the air vesicles, the walls of which were obliterated. The centers showed large areas of coagulation and necrosis. Outside of this was a zone of organized tubercular tissue, the surrounding air vesicles showed cellular exudate.

Kidneys. The capsule stripped readily. Microscopical section showed considerable hemorrhage into the glomeruli, tubes and interstitial tissue, acute congestion but no tubercles.

The heart, liver, intestines, and spleen were negative.

The man evidently died of miliary tuberculosis of lungs and probably of the meninges as well as it is more than likely that there were miliary tubercles in the pia. These are difficult to determine unless the membrane can be held up to the light when they appear as little dots. If postmortems were held on more of our cases, I wonder if in many instances the death certificate might not be changed or at least modified.

## DISCUSSION.

**Dr. Eagleton,** Newark, N. J.: I want to speak because Dr. Richards spoke as if there was nothing found to account for the symptoms. We have found that tubercular meningitis is very deceptive. There may be no suggestive appearance, but if you will take the pararachnoid and hold it up to the light you will see numerous tubercular spots. Many deaths from tubercular meningitis have not been recognized even at post mortem because of the failure to recognize that miliary tubercle may be scattered through the pia, and not recognized by the naked eye. If the part is torn loose and held up to the light it is easy to see the little spots. These occur generally around the base. There is another pathological condition that occurs in life but is not found at death. If you make a large bone-flap over a man's skull—a man that has been suffering from mental disturbances—you will find, during life, that the pia arachnoid is full of a little fluid looking like scales of fish. This can be evacuated by touching with a needle, and a large amount of the fluid comes out. But after the man dies nothing is to be found at post mortem. The fluid has gone back into the lymphatic space and the appearances are normal. So I should say that Dr. Richards' case probably had fine miliary tubercles that would irritate the trigeminal, and possibly had an accumulation of cerebrospinal fluid irritating this space—that would not be discovered post mortem.

**Dr. Worthington,** Baltimore, Md.: I will tell you of a sad case that occurred to me once. Some years ago I removed the tonsils from a young man, a dentist. He stated that his "colds" were so constant as to interfere with his practice. His lungs were gone over well and he went through the routine examination, with negative results. The operation was uneventful. The next morning I found him sitting up in bed and wishing to go home. I advised him to stay until afternoon. His lips looked a little blue but he had no cough, or other suggestive symptoms. I advised him to stay until the next morning, and he consented reluctantly. That evening I was called to the hospital to see him. He was sitting up in bed, his lips were quite blue. I ordered oxygen, and this cleared up. After that he rapidly grew worse. I had several men to see him, but there were no indications of pneumonia, although there was a slight subcrepitant rale on expiration, over several areas in the lung. In a few hours he began to expectorate freely and the expectoration contained myriads of tubercle bacilli. The man died of a miliary tuberculosis that had been latent, although everything had been done to exclude it.

**Dr. Matthews,** Rochester, Minn.: It has been our experience lately that the people themselves want autopsies. We find that the more we get the more the people want. The public are getting so that they ask

for the autopsy of their own accord. I think it is desired in 90 per cent. of the private cases.

**Dr. Richards**, closing: I think we did see a few tubercles beside one of the fissures. Another thing is the fact that you do miss the miliary tubercles after physical examination. I do not recall that this patient coughed at all. I reported this case for the same reason that I wish to congratulate Dr. Worthington on his; because it is finished, and so much of the time you must know the finish of the case to come to a definite decision. We should do in our hospitals what I understand Dr. Matthews does in his: ask for autopsy in every case, not for the benefit of the doctor but for the benefit of the people, for the relatives. Such knowledge is necessary for life insurance, and for other important reasons. If we could only impress it upon the relatives that such investigation is for their benefit; not ours, then autopsies would be more frequent and our knowledge more accurate.

# FIBROMA OF THE LEFT NASAL CHAMBER, WITH EXTENSION INTO THE POSTNASAL FOSSA. REPORT OF A CASE.

By THOMAS CHEW WORTHINGTON, M. D., Baltimore, Md.

There is little known as to the etiology of nasal fibroma. This particular form of nasal or nasopharyngeal tumor is more frequent among males: with comparatively few exceptions it is confined to youth and early adult life. As the name implies, fibroma is "a tumor composed mainly of fibrous or fully developed connective tissue." (Dorland).

Bosworth[1] says: "Perhaps no form of neoplasm develops and spreads with a more unrelenting progress than a fibroma; neither tissue, cartilage nor bone seems to offer the slightest hindrance to its growth."—On account of its location and its nearness to vital parts, a fibroma of the nose will eventually prove fatal, unless subjected to successful operative interference."

According to Price-Brown[2] of Toronto: "The relentless growth of fibroma is one of its characteristics, and in this it resembles sarcoma.

The more irregular contour with the presence of greater pain and a larger amount of surface sloughing should distinguish the latter; but it will require microscopical examination to complete the diagnosis.—Without successful operation the result will always be unfavorable,—a fatal result is inevitable."

Parker[3] of London states: "The symptoms are gradually increasing nasal obstruction, mucopurulent and later purulent and blood stained discharge, attacks of severe epistaxis, a slowly progressing deformity of the nose and face, headache, anosmia, and deafness.—In appearance the surface of the growth is smooth and red, but often coarsely lobulated. It is hard and immovable and highly resistent to pressure with a probe.—If left alone it causes death by exhaustion, or encroachment on vital structures. —The only successful treatment is the complete removal of the growth, the exact method of accomplishing which will depend on its size and attachments."

1. Diseases of the Nose and Throat, 1897, p. 227.
2. Diseases of the Nose and Throat, 1900, p. 129.
3. Diseases of the Nose and Throat, 1906, p. 369.

In the recent work of Wright and Smith[4] we find the following: "Personally Dr. Wright has seen only two or three growths, in twenty years' experience largely devoted to histological work, which he has been disposed to call fibroma of the nasal chambers proper. Such purely intranasal growths as show little or nothing, but dense wavy fibers of connective tissue with sparse nuclei or cellular elements, and scanty blood vessels are exceedingly rare; round, smooth, hard, their density is such as to distinguish them from the usual nasal growth; their base of attachment is usually a more or less constricted one; their removal is usually not followed by recurrence."

J. Bland-Sutton[5] says: "Fibromata or tumors composed of fibrous tissue were regarded as common, but careful histological research has shown them to be very rare."

The abstracts which I have just read, have been taken from well known authors and definitely indicate the following opinions; that true fibroma is a rare form of nasal growth, and that sooner or later nasal or nasopharyngeal fibroma will become incompatible with life, either from pressure on important structures, or from the exhaustion of hemorrhage incident to this disease. It is a clinically established fact, that with few exceptions fibroma of the nose, the nasopharynx, or both is sessile; its base of attachment is broad; when pedunculated the question as to whether it is true fibroma is in doubt until verified by the microscope. The treatment of nasal fibroma is surgical, whether by the knife, forceps, snare, cautery, or by the injection of destructive chemicals. The application of radium and of the X-ray have as yet been limited. The position of the tumor near important structures, its density, broad attachments and vascular environment make strong efforts at evulsion dangerous in the extreme, yet this danger is not nearly so great as that resulting from the severe and exhausting hemorrhages which sooner or later will occur; but which rapidly supervene on surgical interference with these fibrous tumors. The prognosis is bad in proportion to the size and attachments of the tumor; that the ingenuity of the surgeon is often severely taxed, I found to be true in the case which I now desire to report:

R. D., white, aged 16 years, was referred by Dr. J. E. Bromwell of Mt. Airy, Md., June 17th, 1902.

*History.* He does not remember ever having breathed well through his nose. For about one year the left side of the nose had been entirely closed; very little air passes through the right side and then only at times. There is diffi-

4. Diseases of the Nose and Throat by Wright and Smith, 1914, p. 247.
5. Tumours Innocent and Malignant, 1906, p. 59.

culty in swallowing. There is a confused feeling in the head. He cannot think well.

*Examination at office.* Present condition and description of the nasal growth: The right nasal chamber is very narrow; this is caused by pressure on the septum from the mass in the left nasal chamber. Anteriorly the fibroma fills the left nasal chamber completely, and protrudes from the vestibule of the nose. The external nasal wall and the septum are eroded. The inferior turbinal body has been destroyed. The appearance of the growth is a grayish pink; it is firm to the touch and immovable. No air passes through the left side of the nose even with the greatest effort, and very little air passes through the right side. Posteriorly the left choanal opening is sealed; the growth extends from the roof of the postnasal fossa of the left side to the palatal floor, and across to the right side: in a pyramidal form it arises from the floor of the fossa and partly closes the right choanal opening.

Subsequent operative measures showed: that the fibroma was attached broadly to the body of the sphenoid, including the sphenoidal sinus which had been

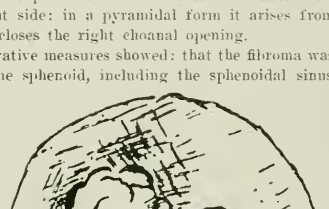


Fig. 1. June 17, 1902, before operation.

completely obliterated, and that the ethmoidal labyrinth had been greatly destroyed.

The following examinations and subsequent operations, were made at the Baltimore Eye, Ear and Throat Hospital:

June 18th, 1902—Examination under cocain and adrenalin. No impression could be made on the growth either anteriorly or posteriorly. Even with slight effort at examination bleeding was profuse but was easily controlled.

July 12th, 1902—First operation under general anesthesia. In order to guard against excessive hemorrhage incident to operation, I made use of Bernays' large aseptic sponge in the following manner: Linen tape was fastened to the sponge through two openings made in the center of the disc, and after the patient was asleep the ends of the tape were carried up through the right side of the nose, and given in charge of an assistant. The Bernays' sponge was kept well out of the mouth and protected by the assistant. The instant it became necessary to stop the hemorrhage the sponge was pushed up into the postnasal fossa and by drawing forcibly on the tape the fossa was tightly closed. The pressure was equalized by the swelling of the sponge; thus immediately checking all bleeding posteriorly. Anteriorly the hemorrhage was

controlled by the usual methods. There was no postoperative hemorrhage at any time.

The snare could not be used either anteriorly or posteriorly.

*Cutting or evulsion* forceps made no impression on the growth. I made use of Asch's gouge within the nose in the effort to break through or to loosen the growth from its attachments; apparently with some effect, but in a very few minutes much blood was lost and it quickly became necessary to tampon tightly.

July 20th, 1902—A stream of water could be forced through the left nasal cavity, but no air could be forced through the nose by the patient. The growth in the nasal passage was apparently smaller than at the time of the operation on July 12th; the growth was not so full in appearance and viewed in the postnasal mirror several irregular openings could be seen. At this examination July 20th, a partially detached piece of the fibroma was removed by evulsion, through the postnasal fossa. A microscopical section of this piece was made. The boy's breathing was still so unsatisfactory, that he was anxious for another operation.

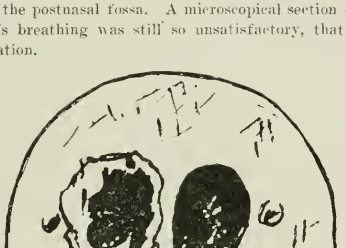


Fig. 2. After September 18, 1902

July 29th, 1902—Second operation under general anesthesia. The growth was still so adherent and unyielding that I hesitated to use the force necessary for evulsion. With long specially made transfixing needles I endeavored to engage a part of the growth with the snare, but with no success.

A Bosworth saw was then forced through the left side of the nose and into the postnasal fossa, and with a finger in the fossa as a guide, I endeavored to saw through or ream out the growth; but on account of profuse bleeding it was again necessary to tampon as before. After the tampon was removed and in order to retain the opening made, and for the purpose of exerting pressure on the growth, a rubber obturator fashioned from a piece of thick gray rubber, was now passed forcibly through the nose, and well back into the nasopharynx; this was worn for two weeks. It was removed only for cleansing purposes during this time.

Sept. 17th, 1902—There was less growth in the nasal chamber except about the sphenoidal region where the fibroma was still prominent; the size of the growth in the postnasal fossa was also less, and the mirror showed several large openings into the nose.

Sept. 18th, 1902—Third operation under general anesthesia; again the snare could not be made use of, the toughness and smoothness of the growth was such that no piece could be caught and held within the loop of wire; therefore alternating with Beckman's scissors and Seiler's knife passed in through the nose, I endeavored to destroy the growth within the nose and around the choanal opening as much and as quickly as possible; this was followed by rapid evulsion with forceps used both anteriorly and posteriorly; by this method several large pieces of the growth were removed. This required only a few moments, but bleeding was severe and the nose and nasopharynx were again tamponed tightly.

After the third operation on Sept. 18th, 1902, the nasal breathing was clear; about the outer margin of the left choana and around the floor, only an irregular rim of the growth could be seen, the growth in the lower part of the right choana was also much smaller; the sphenoidal body was yet well covered with the growth but to a much less extent then before the last operation.

On Dec. 3rd, 1902, I noticed a slight increase in size of the growth about the sphenoidal body extending to the outer wall of the left nasal chamber;

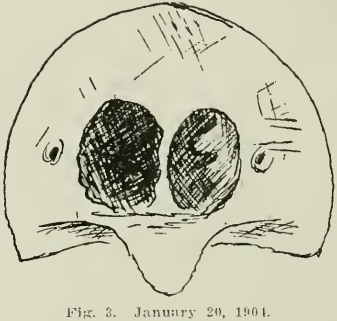


Fig. 3. January 20, 1904.

the patient had lost much blood and he was very anemic; he was in no condition for further operative measures at this time; on this date I therefore made two injections into the growth of a saturated solution of zinc chloride (10m. each); this produced a slough, and for a week or more caused severe pain. This treatment seemed to have impaired the nutrition of the growth temporarily, but in size it soon assumed the same proportions as before the injections.

Between January 1903 and March 1903, I made several injections into the growth; alternating with trichloracetic acid, and with crystallized carbolic acid; the reaction from these injections including pain, stiffness of the jaws and neck, and with an offensive slough, was out of proportion to the diminution in size of the growth. On August 18th, 1903 there was a marked increase in size of the growth about the sphenoidal body and downward into the nose. The patient had not yet recovered his strength from previous operations; Dr. Christian Deetjen by my request now used the X-ray on the growth, applying the ray through a tube in the nose; after four exposures the growth was not so plump or full as before and it was much paler; under the continuation of this treatment the fibroma gradually disappeared, and on January 20th, 1904, no

growth was perceptible in the nose or in the postnasal fossa. A year later on the patient was again examined and showed no sign of the growth; he was then in robust health.

After a period of ten years from the time of disappearance of the growth (March 1914); the patient came to see me; the nose was still clear and there was no evidence of the fibroma; but he was ill; his legs were edematous; he had recently contracted syphillis, and had been a very heavy drinker for several years. A few weeks later his mother informed me that her son had died from disease of the kidneys, at the age of 28.

Dr. Standish McCleary, Professor of Pathology, in the University of Maryland, has kindly prepared the following description of the section, made of a mass of tissue removed from the postnasal fossa, after the second operation on July 20th, 1902:

"Part of the surface is covered by a mucous membrane composed of stratified ciliated epithelium, resting on a tunica propria containing mucous glands. The bulk of the section consists of closely packed bundles of dense fibrous tissue; between the bundles are the characteristic cells of this form of connective tissue;

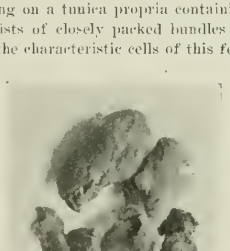


Fig. 4. Specimen.

some of the bundles are hyaline, but nowhere is there any tendency to myxomatous change; the blood vessels are abundant and of considerable size; attached to one margin of the section is some cartilage containing quite an amount of fibrous tissue in the matrix; the growth is a particularly pure form of *fibroma durum*, hard fibroma."

In summarizing I would call attention to the following:

First—That only the most radical treatment could have impaired the vitality of this growth.

Secondly—That notwithstanding the fibroma had been nearly destroyed, it suddenly showed evidence of regrowth about the sphenoid, and that after treatment by the X-ray its absorption became complete.

Thirdly—That until the young man's death ten years later there was no return of the fibroma, and finally that in the case here

reported, the fibroma showed every evidence of endonasal origin in the sphenoidal body, subsequently invading the postnasal fossa by extension.

In conclusion I would point out that few cases of nasal fibroma are reported *as such* although there has been a steady increase in the number of cases reported of nasopharyngeal fibroma.

In the Index Medicus of "Oto-Laryngology" for the years 1909-15 inclusive, thirty-five cases are reported of nasopharyngeal fibroma. We find but one case of edematous fibroma of the nasal fossa, one of choanal fibroma, and one case of fibroma of the nose. This further indicates the rarity of true nasal fibroma, and this as well as the satisfactory result of treatment is my reason for reporting the case.

The specimen shown in the photograph, represents but a small portion of the growth removed. The drawings as indicated by the dates, show the various stages in the disappearance of the tumor.

## DISCUSSION.

**Dr. Kittredge,** Nashua, N. H.: About eighteen years ago I removed a growth that almost completely obstructed the posterior part of the nose. It was about the size of a half dollar, and three-eighths to one-half of an inch in thickness, on the middle meatus posteriorly. I succeeded in getting the wire around it and drew it off slowly. I had no severe hemorrhage back into the nose. Occasionally I see this patient now, but he returns at longer intervals. The growth was pronounced fibroma by the pathologist.

**Dr. Coffin,** New York, N. Y.: These cases are always interesting. Dr. Wells of Washington, D. C., presented probably half a dozen such cases, that he had cured by use of the adenoid forceps. Dr. Harris has suggested that perhaps they all came from the nose. The first one that I know of being injected was one done by Dr. McCullagh. I think he got a complete success, by the use of monchloracetic acid. He had attempted operation, but had fallen down as to courage on account of the hemorrhage. In a case that I saw in New York, similar to this, and which came from the posterior sphenoid, the surgeon had evulsed the tumor and claimed to have a perfectly clear, free nose. He did find fault with the pathologist because he had not given him a definite answer as to whether the specimen was or was not malignant. He had written him that he had "hedged" and the reply came back from the pathologist that he had meant to hedge; that since he had seen a certain case that Dr. H. Smith had (a fibroma that died), he would not give a definite opinion on any amount of a specimen sent him from a case of this type.

**Dr. Chambers,** Jersey City, N. J.: If the doctor had given a description of a case that I have had in hand for six months he could not have described it better than he has now. This morning I have asked Dr. Hershey to help me out in getting a cautery knife to be enclosed in a cover to go in and stop the hemorrhage. I have found that the flow makes a short current with the cautery knife and the hemorrhage stops. I have operated five times in this case and was covered with blood. The boy still stays by me. The X-ray showed the antrum empty

and the tumor confined to the outer wall of the nostril. It is not connected with the sphenoid at all but from the antrum itself. I had an X-ray of the tooth made, feeling that the tumor arose in the antrum of Highmore. The boy feels good. I was going to use this cautery so that the current would be continuous, but I shall use the X-rays now.

**Dr. Harris,** New York, N. Y.: I want to congratulate Dr. Worthington upon the opportunity he had of carrying this case through to a finish, and upon the very careful study he has made of the case, and the complete report. I do not presume to make my memories stand in any way as an exception, or to oppose them to Dr. Worthington's study of the literature, but I understood, when I had some of these cases a few years ago, that a number of the foreign writers were of the conviction that these cases, so-called nasopharyngeal fibroma, had their origin in one of the sinuses. So, if that is true, the cases that have been reported to the profession as nasopharyngeal fibromata were not nasal in origin. Another point in Dr. Worthington's paper is the successful and permanent result that he got. It has been my experience that, in spite of all that we can do, the growth will recur. To quote Dr. Delavan, "It is the natural history of the condition to recur in spite of all that can be done." A case I had ten years ago, which I reported elsewhere, had the disagreeable feature of repeated hemorrhages. In spite of tying up the carotid and taking every other precaution known, on more than one occasion the man nearly bled to death. The growth went into the right antrum, and we found that operative work was of no avail. The best results were obtained with the Coffin attachment to the nasal syringe, and trichloracetic acid with electrolysis through the nose. I am much interested in Dr. Worthington's use of the X-ray. If this could be applied in these cases I believe we should have a wonderful aid in our work which latter, up to the present time, has been discouraging.

**Dr. Mackenty,** New York, N. Y.: Whether this growth is malignant in its histological structure or not, these cases become malignant in many instances. If the growth is sessile and widely attached I don't see much use in attacking it from the surface. The safest means are those that tend to eradicate malignancy; i. e., an operation to attack it at its base. This cannot be done through the pharynx without removing a great deal of tissue. I have seen only a few of these cases, but in those I have seen I believe that, if taken early before they had reached an extensive condition, it would not be such a difficult matter to enter the nose by removing the anterior wall of the antrum and getting behind the growth. I think that chopping it off, or cutting out pieces do very little good. The pedunculated growth is much less malignant than the sessile.

**Dr. Matthews,** Rochester, Minn.: I have seen a number of these cases, and almost all of them had been operated upon before I saw them, with one exception a growth that sprang from the posterior end of the middle turbinate and was a pure fibroma. We removed this and cauterized the whole region as deeply as we dared with the electric cautery. The case remained well. Another had extensive growth under the antrum—a tumor as large as a walnut, with swelling of the cheek. This was taken out and the case remained well. Six or eight other cases that I have seen have resisted whatever measures we employed, and at least two have had a definite diagnosis of simple fibroma becoming fibrosarcoma. In the last four or five cases we have removed the mass and then used radium. As to the hemorrhage, that has been controlled by the radium. I have not applied the radium myself, but I think it is applied through the nostril and nasopharnyx, using three different tubes. I know that not only is the growth itself controlled, but the hemorrhage also, by the use of radium.

**Dr. Richards**, Fall River, Mass.: I ran across one such case in a young man, who, after the removal of the superior portion of the growth, further operation being prevented by the hemorrhage, disappeared without a cure and came under the care of a Boston specialist. I understand the diagnosis was fibrosarcoma, and the patient died. The use of the X-ray in Dr. Worthington's case was quite wonderful in the fact that it did accomplish something, and the young man died of a different trouble.

**Dr. Worthington**, closing: I judged that these growths began in the nose, but when we see them in our cases they are usually far away. Fortunately, my patient came to me before he had such a tremendous nasopharyngeal outgrowth. It is the only case I have had. I looked over all the literature that I could find and learned that the men who had done the most in this line seemed to feel that after early middle life, if the patient could be kept alive, the growth could be resected. My effort was to destroy the growth; to remove as much as possible and with the Bernays' disc control the posterior hemorrhage. The man bled pretty badly and I had to stop it quickly. I do not believe that the X-ray would control this condition because he was closed up tight. When he was getting some air through partial removal of the growth radium would probably do good. I have seen only one other case of this character, a man who died on the table. The doctor, a capable man, had taken all precautions, but the patient died.

# A PLEA FOR THE INCLUSION OF NECK SURGERY IN LARYNGOLOGY.

By JOHN EDMUND MacKENTY, M. D., New York, N. Y.

It is with some hesitancy and after several years of deliberation that I come before this section to present a subject for your consideration on which I know there is a strong diversity of opinion. I believe that the majority of the throat specialists would disagree with me and adduce good arguments to support their stand. Even in New York, the Mecca of indifference, where one may pursue almost any bent unnoticed, I, and others, some of whom I have encouraged to take up this larger work, have not escaped the criticism of the general surgeon. In the smaller centers this barrier must loom larger, and act as a material deterrent to those who might wish to cross the lines so long fixed by custom, and take up the surgery of the neck. After many years of personal experience and observation of the work done by the general surgeon and the throat specialist in this field, I have come to the firm conviction that the surgery of the neck should be looked upon as a special branch of surgery. It is just as much an entity as abdominal surgery or gynecology. To qualify for it, special study in anatomy, pathology and diagnosis is required with an abundance of clinical material on which to build experience. The neck presents many problems and conditions not covered by a general surgical experience. This is not sufficiently realized by either the laryngologist or the general surgeon.

The neck is the most concentrated anatomical field in the body. An indifferent knowledge of anatomy may serve in other surgical fields, to turn out very creditable work, but not so in the neck. Here one must know the relations from all angles and not be led astray by pathological distortions. In deep, widespread neck infections we all know how these anatomical relations may be obscured and lead to an inadequate operation, wherein the dangerous paths into the mediastina may be overlooked. No half measures will serve here where nature, hemmed in by dense fascial coverings, can but poorly help herself in bringing her septic load to the surface. So great may be the inflammatory

edema that both courage and knowledge are required to carry the operation to its proper conclusion; that is, the liberal opening of all the deeper planes as far as the prevertebral fascia. Tuberculous glands infiltrating and extending from the cranial base to the apex of the lung, surrounding and following the deep vessels behind the sternoclavicular junction present to the surgeon as formidable a task as well can be imagined. I pick these two conditions out of many equally difficult, to emphasize the serious nature of this work. The special skill and experience required for its best performance should argue for its holding a special place of its own in surgery.

The tendency today in general surgery is, so far as I can observe, towards rapidity of technique, with its consequent traumatism. In anatomical fields less densely crowded than is the neck with important structures, this technique may be conservative of life by shortening the surgeons stay in the highly sensitive zones, such as the abdominal cavity. When the rapid fire surgeon attacks the neck he is in serious danger of injuring important structures. The spinal accessory, the phrenic, the recurrent laryngeal, the cervical branch of the facial, the brachial plexus, the thoracic duct, the subclavian vein, the apex of the pleura, etc., have all played a doleful role in the after results of careless neck surgery. How many general surgeons, outside of the few who have put in much time in the dissecting room, are able to readily locate the thoracic duct in a living subject? How many know that the recurrent laryngeal is by no means constant in its manner of entering the larynx? I do not criticize the general surgeon as my own shortcomings are too many. I simply wish to emphasize the fact that the neck has not been sufficiently studied, and is approached with a cocksureness which the difficulty of its problems do not justify.

Traumatic surgery in the neck may induce shock. I have seen death occur from rough handling of the pneumogastric nerve. Traumatism favors infection, which is readily induced in the neck, resulting, perhaps, in a serious general infection, mediastinal involvement, or at best a tedious convalescence and deforming scars. Therefore, I contend that the neck surgeons should have a finer technique and a more accurate knowledge of the subject, in all its phases, than is possessed by the average general surgeon.

Between the head surgeon (I presume that all are now agreed upon this designation) and the general surgeon lies this

surgical isthmus, the neck. To whom does it belong? But a few years ago the raising of such a question before this Society would have seemed foolish, so surely and strongly was the general surgeon entrenched upon this ground. Fifteen years ago, after nine years in general surgery, I began the study of our specialty. It was with difficulty that my superiors in The Manhattan Hospital were induced to allow me to take up the surgery of the neck. It was then that I began to visualize the greater future of laryngology and to work for its accomplishment. Now there are several men in that same hospital doing very creditable neck work. It does not stretch the memory of any of us to the breaking point to recall the time when rhinology and laryngology could be practiced with a nasal saw, a set of sprays, a few topical applications and a tonsillotomy set. How different the picture is now. Then our confreres in other departments of our noble art poorly concealed their contempt at our feeble strivings. I might here state that they have not yet fully awakened to the wonderful advance we have made, or to the technical difficulties in our path. An appendectomy is a major operation, but our intranasal radical work on the sinuses is only a minor procedure! To one who has done them both and who knows their relative difficulties, this comparison is, to say the least, painful. I trust that you will pardon the foregoing preamble, but the point of it is this: If, in so short a time, we have accomplished so much, is it a vain dream on my part to look over the horizon's rim and see new fields for our endeavor, to hope that, when the shelf claims me, I shall be able to look from that undesirable perch upon many neck specialists drawn from our ranks?

Many pathological conditions which are conceded to be in the domain of laryngology have their secondary out-croppings in the neck. Toxic goitre is, without doubt, frequently due to tonsillar disease and sinus infections. I have known intranasal malignancy to give its first manifestations in the deep glands of the neck, and I have seen these glands removed by the general surgeon without any suspicion on his part, of their origin.

The diagnosis of laryngeal cancer is our responsibility; why not its treatment? If not, we are assuredly in the unsatisfactory position of one who, starting something, is unable to finish it. Many general surgeons have wrestled with this terrible disease but few have persevered or can show creditable results. Crile stands foremost in America, with fewer surgical deaths than any

other general surgeon, due to an unusual technical skill and to the avoidance of traumatism. The two stage operation is the outcome of the general surgeon's failure to make good with the more logical one-stage procedure, which is employed with success by the laryngectomists abroad. The most expert of these, in my judgment, is a laryngologist.

Laryngectomy is rapidly coming to the laryngologist. In my humble opinion this is as it should be. To support this statement I shall relate one isolated experience. Recently I removed a carcinomatous larynx. For two days progress was uneventful. Then, without apparent cause, the breathing became rapid and labored, stupor, cyanosis and a pulse of 160 foreshadowed the approaching end. It occurred to me to look into the lungs with a bronchoscope. Both main bronchi were almost occluded with inspissated secretions. When these were removed the patient soon returned to his former condition.

If laryngectomy is to be conceded to us, we must be prepared in many cases to do a block dissection of the neck. And if we can accomplish this successfully and expertly, we surely are not far removed from the ideal neck surgeon.

More and more the neck cases are coming to our clinics, placing in our hands the very opportunity needed to perfect ourselves in this work.

I do not wish to be understood as advocating that all laryngologists should enter this field. There are many whose inclinations do not urge them in the direction of major surgery, and there are others whose lack of general surgical training prohibit it. But there is a goodly number in our ranks whose early experience, training and innate talent would justify them in working in this direction. To the younger men I would direct my appeal. You must keep the goal constantly in mind, for without an intense visualization of perfect attainment nothing can be accomplished in any department of life. Work, born of desire to succeed, is the only royal road to success. A thorough knowledge of anatomy, learned in the dissecting room and in the morgue, is the foundation on which the superstructure of pathological anatomy and clinical experience must rest. Much time spent in observing the work of others and in following cases through the convalescent period, is absolutely essential, since it enables us to select technical points suited to our needs and keeps us out of the rut into which every surgeon is prone to fall, working by himself.

The new laryngology and rhinology is developing a high class of technical skill far more refined than that required in general surgery. On this point I speak from experience; for, though engaged in the latter for years prior to taking up my present work, I had a long and discouraging struggle before acquiring what little skill I now possess. The intranasal radical on the sinuses eluded my most patient endeavors, and came to me only after spending two years teaching in the morgue. Such technical refinement is a paramount asset in the surgery of the neck.

I am as well aware, as are my critics, of the weak points in my plea that laryngology should include the neck. Many come into our specialty from medical practice and are too old to acquire the necessary surgical knowledge and experience. The special clinics are often but one department of many in a general hospital, where the lines are fixed and where the surgeon guards jealously what custom has long set apart as his. To even look with covetous eyes across this barrier might bring disfavor upon the laryngologist as well as financial loss.

Our present day training of the laryngologist is not conducive to the best and broadest comprehension of the subject. Many take up special training on receiving their degree. Others, more fortunate, have the usual two years in a general hospital, which I regret to say, serves but too often to engender in their minds a false estimate of their knowledge and experience. Major operations seem easy when guided by the hand of the attendant. It is only when working unsupported that one comes to the full realization of one's ignorance and is spurred on to new endeavor.

A third class has both hospital training and individual experience in practice before taking up the study of laryngology. Here we have the best preparation for a broader conception and better execution of our specialty, were it not for the lamentable fact that so many youthful years are consumed in its execution. Under the present time wasting system of preliminary education, he who enters our ranks through this portal is no longer young.

Where then are we to look for our neck surgeon? It is my belief that we must turn to those who have by good luck, design or lack of financial means escaped the conventional system of education (at least in some of its parts) prescribed by our universities and whose minds have, by force or chance, been focussed early upon their life work.

There are many such men in our ranks who, with hospital internship, general surgical experience in practice, and special

training behind them, are still young enough to eventually qualify as neck surgeons as well as laryngologists.

If criticism you have for this feeble plea of mine, allow me to state, in my defence, that it is of the future I speak. Of our achievements in the past twenty years, we may well be proud and justified in the prophesy of greater things to come. My hope is that laryngology will, eventually include the surgery of the neck, and thus be lifted to a higher plane of endeavor, bringing greater credit to the most progressive specialty of them all, rhinology and laryngology.

## DISCUSSION.

**Dr. MacCuen Smith**, Philadelphia, Pa.: I think that this is not only a very important subject for us to consider, but I believe it is very important for the medical colleges to consider it also. From my experience in this city I think there is a general tendency, and a very decided one, to take away from the laryngologist and otologist as much as they possibly can. So I am very glad to hear this paper, and certainly support it, and believe that this work (surgery of the neck) should be done by the laryngologist. The general surgeon is very frank to admit that these special branches have been perfected by the specialist. Recently one of the general surgeons in this city stood throughout a radical mastoid operation and then came to me to say that he had never before realized what such an operation really meant, and that he realized the bungling he had done in the few operations that had come under his care. I think, in this city, we shall have great difficulty in taking the neck work away from the general surgeon because he is so thoroughly entrenched. We do not have so much difficulty in ear work. I should be very glad if the author of this paper would send a reprint of it broadcast so that it may fall into the hands of the profession at large.

**Dr. Harris**: I want to add my very hearty commendation to what has been said. There is one part to which I wish to refer: In regard to his criticism of our present method of medical education. I thought Dr. MacCuen Smith would speak of it, because he has been interested in this subject. One reason why we have not gone into brain surgery or surgery of the neck is not because the general surgeon has kept us out, but that we have not been fully competent. It is among men who are teachers, and who have gained a sound medical education that, with special training in anatomy and pathology are able to do what Dr. MacKenty has done, who has had nine years in general surgery. But I firmly believe that it is possible for the young man to get that training, if he will, although it is perfectly true that the average man that goes into otolaryngology goes into it without a good training in surgery and physiology, and is not competent to do the work. If he does go into general surgery he lays us open to the criticism that we do do not know the simple principles of asepsis.

**Dr. J. Solis Cohen**, Philadelphia, Pa.: I have always contended that the laryngologist should do his own surgery, and his tracheotomy and surgery upon the thyroid gland, and I am very glad to see that it is generally admitted to be the proper thing. It used to be the fashion that when the laryngologist laid aside the atomizer he handed the knife to the general surgeon, but I am glad to say that this is no longer the case.

**Dr. McCallagh:** The subject of this paper is of great interest to us all. I believe that the thing to do is to have the medical schools adopt a post-graduate course of, say, two years, so that a man who wishes to train himself as a specialist would thus have an opportunity. A great many of our specialists admit that they do (not) have to prove that they are specialists. Anyone who sees some of the men who, after six weeks to three months training go to the smaller cities and pose as specialists, will realize the need of more extensive training.

**Dr. MacKenty** (closing): I thank Dr. Dench for his words of encouragement from the general surgeon. I trust they will continue to look upon us with favor. It is a good sign. There seemed to be a little misunderstanding about what I meant by preliminary education. I meant that a man's training should be so focussed through his preliminary studies that when he is through with them he can get at the real work that he is going to do in life before he is too old. I have a boy, now going through school, who, if he follows the regular course of training, will begin the practice of medicine when he is thirty. That is too old. The colleges should have some course by which the expectant doctor can get the greatest amount of help in the work he is going to do, having several years of full time, instead of wasting these years in learning a lot of stuff that will be of no use to him in his profession. I thank Dr. Smith for his support. As I said before, I think the general surgeons do not realize our troubles, and I also believe very firmly that brain work belongs to the ear specialist. The best work has been done by the ear-man, and it is for him to develop that end of the work. I am glad to see that you are all recognizing the fact that we have done great things in the past, with progression along scientific lines in our work. I came into this work when it was crude and undeveloped, and I have seen it grow and advance wonderfully. In the beginning it was almost a disgrace to say you were a laryngologist, and the general man looked upon us as a set of "spray-ers"; but now we have reason to be proud of our specialty.

# REPORT OF THREE CASES OF BRAIN ABSCESS OF OTITIC ORIGIN.

By GEORGE MORRISON COATES, M. D., Philadelphia, Pa.

1. Martin O'Connor, 51 years, a waiter, came to the Out-Patient Department of the Pennsylvania Hospital, July 6th, 1912, complaining of pain in the right ear of two days' duration. He gave a history of having had a right sided aural discharge for several days, four months previously, but with no discomfort in the interim. Examination showed a bulging membrane, which was at once incised in the posterior portion of the membrana, a somewhat scanty discharge resulting. Some mastoid tenderness was present which became slightly more marked in the next few days and he was admitted to the Ward on July 9th. He did not complain particularly of headache. No signs of meningitis were present and the leucocyte count was practically normal. The patient was a well developed and well nourished man. Eyes showed no spontaneous nystagmus, pupils were equal and reacted to light and accommodation, eye grounds negative. There was no aphasia. The teeth were in bad condition, throat normal and tongue slightly coated. Heart, lungs and extremities normal. Urine normal. Temperature 99°, pulse 88, respirations slightly high.

Expectant treatment was carried out for a few days, as the temperature continued low, but some slight swelling appearing over the mastoid, he was operated upon on July 15th. The mastoid cells were all found normal with the exception of the antrum, the tegmen of which appeared soft, if not necrotic, this softened area extending outward into the temporal ridge. The dura was exposed for inspection and appeared to be under considerable pressure so that it was incised and the brain searched with a thin knife blade. In a direction inward and upward, about an inch from the surface an abscess cavity containing about two drams of pus was encountered. It appeared to have rather firm walls. Gentle washing cleaned out the pus, a cigarette drain was inserted, and the lower portion of the skin wound closed. Cultures from the pus failed to show any growth. Recovery was slow but perfectly uneventful, the abscess continuing to drain nicely, although the middle ear ceased at once to discharge. The drain was removed in about ten days and the wound allowed to close by granulation, which it did completely in about six weeks. During this time the temperature never rose above 99 4/5°, being normal for the most part, the pulse ranging from 64 to 88. Respirations were always a little rapid.

This case showed nothing clinically to suggest brain abscess, the bulging of the brain and dura through the operative bone wound being the only indication which lead to its discovery. As the contained pus was apparently sterile and as the cavity had definite walls, it is probable that it had its inception at the time of the first ear trouble four months earlier. It illustrates very well the type of abscess that is favorable to recovery, namely a walled off, chronic process with sterile pus or at least having organisms of low vitality, discovered at the time of operation without having given rise to pressure symptoms.

2. Carmen Centrali, 12 years old, school boy, admitted to the Pennsylvania Hospital May 24th, 1913. Temperature 99°, pulse 100, respirations 24. He had had a simple mastoidectomy on the right side two years before, followed by a radical on the left side one year later. On admission he gave a history of pain in the right ear for five days accompanied by a free discharge of pus. For two days past there had been marked general headache. He was a frail, undersized boy, irrational, whining with considerable photophobia. The eyes reacted well to light and accommodation; grounds apparently normal. Heart and lungs were negative. The neck was quite rigid, suggestive Kernig signs were present on both sides and a doubtful Babinski, reflexes not markedly increased. There was swelling and redness over the right mastoid region which was markedly tender. The right membrana showed a large central perforation in a thickened drum, freely discharging foul pus with a distinct musty odor. The left ear, on which a radical operation had been performed one year before was not entirely dry. The leucocyte count was 21,700 with a high percentage of polymorphonuclears. By lumbar puncture the spinal fluid was found to be cloudy and under considerable tension, with the polymorphonuclears 67%. No tubercle bacilli were found and no growth was obtained on culture media.

Although all his symptoms were considerably relieved by the lumber puncture, he was operated upon by Dr. Francis R. Packard, on whose service he was, and much destruction of bone in the upper portion of the mastoid found, extending into the temporal ridge and zygomatic cells. The dura was freely exposed and the wound packed with iodoform gauze. Whereas he had been markedly restless, delirious and noisy previous to operation, he now became much quieter and mentally more clear. Very foul pus continued to drain from the wound, and at this stage Dr. Packard went off duty and he came into my hands. The improvement following the first operation was not permanent, however, and during the next few days all of his former symptoms returned in greater force, the neck being well arched backward and his delirium increasing. On July 9th, ten days after the first operation, in dressing the wound, I noticed that the dura was tense and bulging through the bony opening through the site of the temporal ridge, the removed bone covering an area about the size of a quarter. A paralysis of the external rectus muscle of the right eye was now apparent. The pus was very foul in odor. With very light anesthesia, I incised the dura and searched the underlying brain with a grooved director when a large abscess cavity was discovered containing about an ounce of thin, foul pus. A rubber drain was inserted without washing and the wound dressed. The patient was very weak and partly comatose. The pus showed the colon bacillus in pure culture, and continued to drain very freely, and always foul, until, gradually sinking, he died five days later. At this time there was considerable protrusion of the brain tissue into the wound, the protruded portion being necrotic. The temperature had ranged from 99° on admission to 104° on May 26th where it stayed for two days. It dropped to 99° for a few days and again rose to 104 3/5° when the second operation was performed. Thereafter 103° was the lowest temperature until death. The pulse rate was commensurate with the temperature, running from 100 on admission to 160 shortly before death. Respirations were always rapid—from 24 to 32 and 52 at death. No autopsy was obtainable, but from the appearances presented, I think it probable that there was a second abscess

that was not evacuated. The patient's condition while upon the operating table was so grave that I did not feel inclined to search further after draining the abscess.

3. Laura Hayden, 30 years old, stenographer, was admitted to the Pennsylvania Hospital, July 6th, 1916, complaining of pain in the left ear, left sided headache and general malaise. Temperature 102°, pulse 92 and respirations 24. Family history was without interest. She had had measels in childhood which did not affect the ears as far as she could remember, had always had "catarrh," indigestion and constipation. The left ear had suppurated, with severe earache, fifteen years previously, and had ceased discharging after a few days. During the next few years she had had several similar attacks in the left ear, and finally the discharge had been almost continuous for the past six years although always scanty. She had not had the ear treated, considering it a negligible matter except for the impaired hearing.

The present illness dated back six days when she developed an acute rhinitis from sitting in front of an electric fan while at work. Earache immediately followed and she went to bed. There was an attack of vomiting the same evening and as she felt badly she was admitted to another hospital the next day, July 4th. Not getting any better here and having almost continuous nausea and vomiting she left this institution and was admitted to the Pennsylvania Hospital where the drum was incised at once. Temperature on admission was 102°, pulse 92, respirations 24. She was a well developed and well nourished, full faced, rather pale, adult, complaining bitterly of pain in the left ear. There was no adenopathy, the eyes were slightly prominent, pupils widely dilated, equal, reacted normally to light, grounds normal. The nasal septum was deviated to the left, the teeth in fair condition, tongue coated, throat negative. A scanty discharge came from the left ear and there was no retraction of the head although slight pain on extreme flexion of the neck and a suggestion of Kernigs sign in the right leg. Reflexes normal. No spontaneous nystagmus was present, vestibular reaction to the caloric test normal. Widal and Wassermann reactions negative. The blood count showed a leucocytosis of 14,000, polymorphonuclears 70%. Urine negative. Patient was very uncomfortable. No aphasia—mentally slightly sluggish.

On July 8th complained of considerable pain in the ear and back of the head, as well as in the nose. There was very little aural discharge. The eye examination by Dr. Wm. T. Shoemaker follows: Externally nothing noticably abnormal. Pupils large and equal. Irides react to light. Ocular movements full. Eyegrounds: media clear, discs well defined, vessels good, no hemorrhage or exudation or any evidence of intracranial trouble. Temperature 99°, pulse 80, respirations 20. Her condition continued no worse for two days when the temperature rose to 103°, pulse 84, respirations 20, and she vomited a grassy green substance. Pain was more intense on the vertex and occiput, there was slight rigidity of the neck and Kernigs sign was positive in the right leg. Dr. Morris J. Lewis, the internist, confirmed the diagnosis of meningitis. The mastoid was not markedly tender and there was no sagging of the posterosuperior canal wall. By lumbar puncture 35 c. c. of fluid under pressure were withdrawn. Fluid was cloudy, there was slight reduction of Fehlings solution, cytological count was 186, polymorphonuclears 80%. The blood culture was negative. As the patient was much more comfortable following this, it was repeated the following day, July 11th, and 20 c. c. of cloudy fluid, not under

pressure, were withdrawn, with apparently increased comfort. At midnight, however, she again complained of great pain, which morphia failed to control. She became weak and cyanotic during the remainder of the night and early morning. The pulse was very weak and the temperature went to 104 1/5°, pulse 92, respirations 24. The report of the X-ray taken July 10th was very misleading as it turned out. It gave "extensive necrosis of the entire cell area of the left mastoid, sinus probably uncovered. No further evidence as to pressure or location of any suspected brain abscess."

The left mastoid was opened July 12th and found to be eburnated without cells or necrosis, solid and intact and the kind frequently seen accompanying prolonged middle ear suppuration. Believing that we were dealing with a case of purulent meningitis with the possibility of brain abscess, I freely exposed and incised the dura of the temporal lobe, obtaining a little cloudy cerebrospinal fluid. The tension of the brain appeared to me to be increased so I searched it cautiously with a grooved director and tapped an abscess cavity containing nearly an ounce of thick pus. Up to this time the patient's condition had been precarious. The temperature was 102°, pulse 80 and respirations 28 just before being placed on the table; she was comatose, cyanotic and breathing was slightly stertorus. The pulse was very weak. Very little ether was used in anesthetization. After draining the abscess, almost immediately her pulse improved, her color became normal and her breathing was easier. A rubber drain was inserted, the wound packed and dressed, and she was sent to the recovery ward, by this time already conscious and apparently rational. Before I had finished dressing, however, word came that she was sinking rapidly and when I reached her a moment later, she was cyanotic, although the pulse was fair and continued so for several moments. Respiration became weak and ceased. Artificial respiration and stimulants were of no avail and she died of respiratory failure.

The autopsy diagnosis was multiple (2) brain abscess in the left temporal lobe, purulent meningitis. The complete autopsy report follows:

"There is an operation wound with a drain inserted reaching into the cranial cavity behind the left ear. There is a slight clouding beneath the pia. The various arachnoid spaces contain cloudy fluid. The fluid in both ventricles is purulent—less so in the right. The right cerebrum is otherwise normal. The drain reaches into an abscess cavity within the left temporal lobe, about 3 c. m. posterior to its anterior end and opening in about the line of the inferior temporal sulcus. This cavity is just beneath the surface, measures about 2 c. m. in diameter, contains some purulent fluid and has a soft, thin wall of necrotic cerebral tissue. There is also another abscess which opens into the posterior descending horn of the left ventricle and to the surface proximal to the cerebellum at about the posterior third of the collateral fissure and slightly lateral to that in the fusiform gyrus. This abscess cavity is the larger of the two, in that its openings in both directions are large. There is about 1 c. m. of cerebral tissue in between the two cavities.

*Microscopical Examination*—The edge of the brain cavity is densely infiltrated with leucocytes and to a less extent with plasma cells. There is no fibrous reaction about the inflammatory area. The cavity is not of long-standing."

From the autopsy report these abscess cavities seem to have been of recent formation, and there is nothing in the history to indicate prolonged intra-

cranial involvment except the duration of the aural discharge, fifteen years. Of course, we know that a brain abscess frequently lies dormant for long periods of time to eventually become active and dangerous under some fresh stimulation, such as the chilling from the electric fan and consequent aural reinfection in this case. Although we suspected brain abscess almost from the start from the character of the headache, nothing confirmatory of this could be discovered, and the development of the meningitis was so evident, that that exclusive diagnosis was made. I could not be quite content with this, however, or feel that it accounted for all of her symptoms and therefore searched the brain at the time of operation with the discovery of the first abscess. I did not suspect the second one. Immediately following the evacuation of the first abscess her condition was in all respects so satisfactory that I feel that I was justified in believing that the source of the trouble had been discovered and eradicated, and I sent her away from the table feeling that recovery was not at all to be despaired of. Whether the sudden collapse that followed within ten minutes was due to the rupture of the deeper abscess into the ventricle I do not, of course, know but think that is probably what occurred since such a rupture was discovered at autopsy. Or it is possible that there had been a slow leakage there which accounted for the infection of the cerebrospinal fluid. There was no "stalk" or ascertainable avenue of infection to or from the middle ear, although this must undoubtedly have been the origin of the brain lesion.

These three cases only serve to emphasize still more the well-established dictum that a chronic discharging ear, no matter how trivial it may appear or how quiescent during long periods of time, is always a serious menace to the life of the patient. No one fact pertaining to suppurative otitis media is, perhaps, better established, and yet how many such middle-ear conditions are neglected and temporized with. J. W. Murphy lays stress upon this point in his article read before the Society in June, 1915, and considers cerebral abscess of otitic origin almost always an acute process, and that therefore eye pressure symptoms have not time to develop as in the more slowly growing tumors. In only one of my cases was there any paralysis apparent—that of one of the eye muscles in the second case, and this came on late in the disease. Neither were the mental symptoms more conclusive or helpful in establishing the diagnosis. In one of these cases, the one that recovered, there was little elevation of temperature, but in the other two the febrile reaction was marked; none was ever subnormal. The pulse rate was rapid, at least commensurate with the temperature, as were also the respirations. With wakefulness and delirium rather than slow cerebration, and mental alertness instead of drowsiness, the absence of eye pressure symptoms or of paralysis, and in two of the three cases a commitant meningitis existing, the diagnosis of a brain abscess was a difficult task. As

a matter of fact, it was only made on the operating table after a search of the brain on suspicion, in two of the cases.

## DISCUSSION.

**Dr. Dench,** Philadelphia, Pa.: There is one point that I should like to mention here, and that is, that in the earlier operations upon brain abscess, I am certain that most of us lost a number of cases by meddlesome surgery. In other words, whenever we opened a brain abscess we felt that it was necessary to explore the entire abscess cavity, and as the result of this exploration we frequently spread infection to previously healthy brain tissue. In treating a brain abscess, we must remember that the patient is suffering from two conditions: First, the general infection, and second, increased intracranial pressure. The critical symptoms are usually caused by the increased intracranial pressure, and this increased pressure has existed for a considerable time previous to the operation. If now the abscess is found and evacuated this increased intracranial pressure is relieved, and by the evacuaton of the abscess the focus of infection is drained. My procedure at the present time is to evacuate the abscess freely and then without exploring the cavity at all, either by the finger or by inspection through an encephaloscope, to place a cigarette drain in the abscess cavity and apply a dressing. In this way the increased intracranial pressure is relieved, and the abscess cavity is gradually obliterated by the collapse of the brain substance into the cavity. The parts should be manipulated as little as possible at subsequent dressings, provided the patient is doing well. If symptoms point to a pocket of pus in the brain substance which has not been thoroughly drained by the first operation, then the abscess cavity must be investigated either by the finger or through the encephaloscope. In the absence of such symptoms, however, it is the best practice to simply drain the abscess and insert a drain, and at the subsequent dressings to disturb the parts as little as possible, simply removing the cigarette or other drain and replacing it. In the absence of localizing symptoms, it is better to relieve the intracranial pressure by a decompression operation, to wall off a certain area of the exposed brain by means of gauze packing, thus causing an obliteration of the arachnoid space, and then in the course of 12-48 hours to explore the brain substance. This is the procedure which should be adopted in cases where there are no localizing symptoms, or where the appearance of the exposed dura gives no indication of pus in the brain substance immediately adjacent to the exposed area.

**Dr. Smith:** After all we have to fall back on the very doubtful point of diagnosis in brain abscess. That is, diagnosis in so far as localization is concerned. We may suspect a brain abscess accurately enough, but the exact location is difficult to determine. So, when we think we have a brain abscess we sometimes hesitate to try to relieve it because we do not know just where to operate. This winter I had in quick succession three cases of meningitis, all of which apparently developed very quickly without previous symptoms. Two of them died in forty-eight hours, and the other in three days. The last case, a girl eight or nine years, previously, had been thrown from her horse, taken to the University Hospital, operated upon by Dr. Edsall, and after a long convalescence, recovered. This winter, about four years after her injury, she was taken suddenly ill, became unconscious, was taken to the hospital with her head drawn back and all the symptoms of meningitis. The spinal fluid when drawn, showed distinctly that she was suffering from meningitis. She died and, fortunately in this one case we got an autopsy, it was found that she had suffered from a brain abscess. There was no doubt that the meningitis was superinduced

on the brain abscess which had probably been latent from the time of the accident. This would incline to the belief that we may have a brain abscess remaining quiescent over quite a long period of time.

**Dr. MacKenzie,** Philadelphia, Pa.: Intracranial complications rarely come singly. Uncomplicated brain abscess, no matter what its location, has a certain group of symptoms that we cannot mistake. The pulse is low, the temperature is low, the intelligence and the respirations are low. This is borne out by the fact that when you operate you find that everything is slowed down. Cases that we frequently meet are complicated with meningitis and sinus thrombosis, and in such cases the pulse, temperature, respirations—everything—are high.

**Dr. Eagleton,** Newark, N. J.: There are a few points that I think are very important. Of all the three cases the only one that recovered was the one on which they started to do a mastoid and accidentally found the brain abscess. If we wait for the classical symptoms of brain abscess to appear we have greatly increased intracranial pressure; we have great destruction of cerebral tissue, and when we operate, if you examine the case you will find you are dealing with almost a hopeless case because of the suppuration at the time of the operation. The cases of brain abscess that recover are the cases in which the patient is in otherwise excellent condition; that give no signs of intracranial pressure at the time of the operation. If we, as otologists, are to operate on these cases, my personal opinion is that we must get away from the custom of going through a dirty field to get into a, perhaps, non-infected brain. I would like to ask Dr. Coates in how many cases he has explored the brain in brain abscess and that have recovered? If he has gone through a mastoid wound? That is a reason why we are afraid, because we have learned that if we do a mastoid and find the dura bulging, and we open that dura, we find that the patient is very apt to develop meningitis. What we want to do is to explore, but to explore through an absolutely *clean* field. If we find nothing we merely close the wound and the patient would run very little risk of a mortality result after operation. But we have got to stay away from the source of the infection, and that is the ear. Another point in this paper is that only in the post mortems did he find communication with the lateral ventricle. This serves to exemplify a point that I have always insisted upon and that is that brain abscesses travel inward if they start subcortically. The majority of brain abscesses are caused by retrograde thrombosis of a vein that passes up into the substance of the brain itself, and develop first inside the cortex. These abscesses, because they are caused by the death of tissue as a result of blocking of one of the terminal veins, always travel toward the lateral ventricle; so any surgeon who explores a brain and fails to find a brain abscess, should close his dura. I feel very strongly about this. I have heard it said that we should leave the dura open because then the pus will come out. It *never* comes out. One more point Dr. Mackenzie has stated that everything is slowed down—pulse, temperature and respiration. I do not think that is entirely true. The pulse is slowed down because of pressure. The temperature, however, is slowed down for some reason that has never been explained. I have spent considerable time trying to find the cause of this. The fact is that as long as the suppuration is inside the brain we have a subnormal temperature. But we should not count on a slow pulse in brain abscess excepting when the brain abscess is very large.

# CONSERVATIVE OPERATION ON NASAL ACCESSORY SINUSES.

By JOHN J. SULLIVAN, JR., M. D., Scranton, Pa.

## Steps in the Operation on the Antrum of Highmore.

1. Cocainize the inferior concha under and above it and also the septum opposite, using the Freer method, followed by 1:5000 adrenalin solution.

2. Infract thoroughly the inferior concha upwards. This gives an excellent view of the lateral wall under the inferior concha.

3. Retaining the speculum, if desired, the entire nasoantral wall can be removed. Everything is within sight of the operator.

4. Remove the wall, using a Whiting No. 2 or 3 long mastoid curet, burr.

5. Entrance with the curet should be about the middle and at the point of the attachment of the bone with the lateral wall.

6. The floor of the sinus is lowered to that of the nose.

7. All fragments of bone and mucous membrane are removed by suitable forceps. A flap of mucous membrane can easily be loosened if the operator sees fit. Personally, I never take the time to do so, it prolongs the operation, and is unnecessary.

8. Wounding the nasal duct anteriorly is avoided by keeping below the attachment of the inferior concha. Avoid the descending palatine canal posteriorly by not cutting into dense bone at the posterior limits of the sinus. One inch anterior-posterior is a fairly large opening and I can usually obtain it.

9. Pack with gauze, either iodoform or bismuth. Remove within twenty-four or forty-eight hours and the sinus is then painted with 50% argyrol in glycerin.

10. Occasionally I rasp away the bone in front of and below the attachment of the inferior concha. This allows easier passage of the irrigation tube. I do not favor continual washing of any cavity. Once a week is enough, even in an acute case, providing there are no symptoms of pus retention (pain, chills, etc.).

Nearly all remain open when thoroughly done. This is left to the judgment of the operator. It would border on the extreme radical to make a permanent opening in an acute case. It is always the height of folly to expect an old, chronic case to improve after a small opening. In the latter cases a recurrence may always be

expected following a cold in the head. In the past six years I have operated on about one hundred and fifty of the above cases.

November 23, 1913, I did my first ethmoid operation without either removal or cutting of the middle concha. Prior to that time I had made the anterior cut into the middle concha before doing so. (In the *Laryngoscope*, February 13th, mention is made of this.)

1. Frontal ethmoid. Cocainize and apply adrenalin every five minutes until anesthesia is complete. Takes usually at least one-half hour.
2. Infract M. C. to septum.
3. Remove uncinate and hillae ethmoidalis.
4. Level off and leave no scraps behind. (Must be clean wound.)
5. Open up frontal sinus with curet, or rasp.
6. After treatment, suction and no interference.

*Complete exenteration of cells into sphenoid.*

1. Infract M. C.—Start at uncinate and work back with Mosher curet or any biting forceps, until sphenoid wall is reached.
2. Press M. C. into ethmoid hole and probe for normal sphenoid opening.
3. Enlarge same and take dimensions of sinus.
4. Press M. C. toward septum and break into sphenoid through posterior ethmoid cells.
5. Connect these openings.
6. M. C. and superior concha are then a thin curtain.
7. Middle concha can be trimmed if necessary; hardly ever so, unless to break down cells within it, *i. e.*, toward ethmoid space.
8. M. T. and wall of post ethmoid packed into ethmoid space or allowed to hang.
9. No scraps of tissue, everything smooth.
10. Gauze packed gently to keep M. C. in place.
11. Some cases which have not been thoroughly cleansed, or large amount of pus present, allow cells to granulate and M. C. to remain in place—or remove, if operator sees fit.

*Advantages.*

1. No primary or secondary M. C. removal.
2. Easier to do.
3. Mucous membrane intact.
4. Olfactory nerves intact, no danger of tearing.
5. Not so dangerous.
6. Less reaction and shock, period of repair less.

However, we must remember that without perfect nasal drainage no operative procedure will clear up in the majority of cases. If a

deflected septum, nasal hypertrophies, adenoids, etc., are present there will be kept up a constant source of irritation and recurring infection that could hardly be cleaned up, even per radical. No one would think of trying to get results in a catarrhal middle-ear condition without cleaning up any great nasal abnormalities, yet a good many of us will go on treating sinus inflammations where the ventilation and drainage of the nose are imperfect, expecting to get results. It is often a case of putting the cart before the horse.

## DISCUSSION.

**Dr. Lee Cohen,** of Baltimore, Md.: In regard to the conservation of the middle turbinate, two cases have recently come to my attention that make me feel that we ought to select our cases in which we leave the middle turbinate intact. If we have a long head with a narrow nose, the leaving of the turbinate is apt to be followed by adhesions that will close off drainage. Two cases, having been operated upon by rhinologists, came back with intense headache because of faulty drainage, and upon opening this up, it was found that the middle turbinate, after having had the ethmoid labyrinth removed had been pushed into the labyrinth and adhesions had formed between the middle turbinate bone and the meatus. Those patients both suffered from intense headaches. The adhesions entirely shut off the proper drainage from the labyrinth.

**Dr. Coffin,** New York, N. Y.: I am interested in anything that relates to the sinuses and am especially glad to hear the doctor's description of the operation on the antrum, and especially to hear that he had operated upon children of five or six years of age, because I have considered what would be the best way to deal with those cases. I have just recently been interested in this especially because, as I have said before this Society when I want to get a really, if I may use the expression snotty-nosed kid I do not look in the nasal wards at all. In our wards at the hospital I said to our X-ray man: "Go in and X-ray those heads in the eye-ward!" and every one of them had diseased antrums. I then wished to get some controls, and had him look for noses that were clean, in the nose-ward. These were found to be perfectly normal, with free sinuses. We have those children under treatment in the eye-ward, but in case the conservative treatment did not succeed I was in doubt just what operative procedure I should use. In regard to the ethmoid operation, I do not believe in it at all. I mean to say, if you operate radically on the ethmoid and go up around the attachment of the middle turbinate, I do not believe it is any good. It is very apt to be diseased and I doubt the expediency of pushing it into the cavity. Lothrop makes an opening from one frontal sinus into the other and takes out a certain amount of the septum between the sinuses. He does not care especially whether or not he closes the frontal duct on the diseased side because he has drainage only through the normal sinus. It is a good operation in some cases. I had a case on which I operated on seven times. Dr. Myles has taken care of the lady from time to time, and about every two years we get a recurrence and pus. We go in there and find somewhere a pocket and wash it out and wash it out. The last time I was to do that operation, the night before came Dr. Lothrop's paper, and I read it and used the operation in that case the next day. I did not have his instruments, but I got in through the septum between the two sinuses. The case has been well ever since. There is no question that she has drainage that will not block up any

more. It is not at all an operation that will take the place of the Killian; you have all these ramifications and deviations, but it is a very good thing to have up your sleeve. Especially in these secondaries.

**Dr. Sullivan**, closing, gave a demonstration on the blackboard "We have operated upon about twenty children in the last year, and about half were ethmoid cases. As for the Killian operation, some years ago we had from six to ten of them a year. We have about one or two a year now. This operation is not intended to take the place of the Killian; it is simply to level the middle turbinate bone instead of removing it."

# RADICAL MASTOID OPERATION WITH PRIMARY SKIN GRAFT. REMARKS MADE IN CONNECTION WITH AN OPERATION PERFORMED AT THE SAME TIME AT JEFFERSON COLLEGE HOSPITAL.

By EDWARD B. DENCH, M. D., New York, N. Y.

Before proceeding with the operation, it may be interesting to say a few words about the possible findings in this case and the various procedures which we shall adopt in view of certain findings, also upon the possible effect of the operation upon the function of the organ. We can lay it down as a general rule that if the hearing is very poor as the result of a suppurative otitis media, that is, if the hearing for the whispered voice is reduced to 3 feet and the tuning fork reaction shows that the perceptive apparatus is not involved, the hearing will probably be improved by the operation. If, on the other hand, we operate upon a patient whose hearing is exceedingly good, that is, if he hears the whispered voice at 10, 15 or 20 feet, the chances are that tympanic exenteration will cause a reduction in the hearing.

Regarding the employment of the primary skin graft in cases of the radical operation for chronic middle ear suppuration, the primary graft can be employed in practically every case, excepting in cases where the sinus or dura is wounded. If the sinus is wounded, or if a perforation is found in the dura of the middle fossa, either as the result of the disease or if such a perforation occurs as the result of instrumentation, the primary graft should not be employed.

In all cases of radical operation it is necessary to enlarge the external auditory meatus, the size of the new meatus to be dependant upon the size of the radical cavity resulting from the operation. The meatus must be large enough to completely aerate the cavity; otherwise there will be maceration of the graft and a certain amount of moisture will always be present in the ear. I prefer to enlarge the meatus by the following method: The meatal flap is cut by inserting a probe-pointed knife with a narrow blade into the external auditory canal, bringing it out through the posterior wound. The blade of the knife is first directed downward and a short incision downward is first made.

The incision is then continued first backward, by turning the blade into the horizontal plane, and then upward, directing the blade into the vertical plane. The backward incision determines the breadth of the meatus, and the vertical incision its heighth. The vertical incision usually stops just below the inferior crus of the antihelix. After the meatus has been enlarged in this way, all cartilage and fibrous tissue is removed from the flap formed by the incision above described and the flap is sutured upward and backward against the raw surface of the anterior flap by means of 10-day chromic gut.

If, in this particular instance, I have to deal with a fully developed mastoid, I shall make a large meatus. If, on the other hand, the mastoid is sclerotic and the antrum small, I shall make a small meatus. Roughly speaking, the meatus should be large enough to admit the little finger of the surgeon. For cosmetic reasons, it is naturally more important in women to preserve a small meatus than to enlarge it unduly.

With reference to the primary graft, the graft is usually taken from the thigh. Several grafts should be cut, so that in case the operator fails to apply the first graft to his satisfaction this may be discarded and a second graft applied without loss of time. The graft should be cut large enough to completely line the radical cavity. It is important in cutting the graft not to perforate it at any point, because, as you will see in the demonstration, the graft is made to apply itself to the very irregular walls of the radical cavity by withdrawing the air from beneath it by means of suction. If a minute hole is present in the graft, the air will be imperfectly removed from beneath it and the graft will naturally fail to fill in the various little irregularities in the walls of the radical cavity. It is frequently possible not only to completely line the bony cavity, but to cover the cut edge of the meatus as well, with a single graft. This, however, is not necessary as two or three or four grafts even may be employed with equally good results. In case more than one graft is used, the grafts are inserted so that their edges overlap. This overlapping does absolutely no harm. The grafts are held in position in the cavity by means of small pledgets of sterile cotton. The pledget holding the graft into the mouth of the Eustachian tube has a piece of silk ligature tied to it because it lies so deeply in the cavity that otherwise its removal is sometimes difficult. If the graft is not large enough to line both the cavity and to cover the cut edge of the meatus, the meatal graft is applied in the following manner.

After the radical cavity has been completely covered by the graft and held in place by pledgets in the manner above described, a second graft is slid into the radical cavity along its posterior border, somewhat overlapping the graft already introduced. This second graft is held in position, that is, is anchored in the radical cavity, by means of a gauze packing, and the remainder of the graft is spread out upon the posterior aspect of the wound, care being taken to see that it lies perfectly flat. The anchoring packing is then introduced so as to completely fill the radical cavity. The graft that has been spread out posteriorly is then grasped at its upper and lower angles by means of thumb forceps and turned forward over the packing. The anterior flap, that is, the auricle with the enlarged meatus formed in the way above described, is then turned backward so that the ear occupies its normal position. Looking through the meatus, the operator can now see the raw surface of the graft as it lies turned forward upon the anchoring packing. Through the meatus, the upper and lower angles of the graft are grasped by the surgeon and his assistant, respectively, and drawn through the enlarged meatus. By this procedure the raw surface of the graft is made to apply itself to the cut margins of the enlarged meatus. A second piece of sterile gauze is introduced through the meatus, holding the graft firmly against the cut edges, and the posterior wound is then closed by means of Michel clamps. This completes the operation.

In the case before me, I make the incision in the usual way. Note that I do not completely clear the mastoid tip, as one should always do in a case of simple mastoid operation, because in cases of chronic suppuration we frequently find the lower portion of the mastoid completely sclerotic. If the tip is cellular, it must be removed, but if it is not cellular it is best to leave it. The disadvantage of completely removing the mastoid tip or even completely denuding it of soft tissues, is that it opens up the cellular spaces of the neck and frequently leads to a gravitation abscess. In this particular case, I find the mastoid small, the bone sclerotic, the antrum low and small. The dura is exposed in the middle cranial fossa. The middle fossa is so low in this case that I shall be obliged to enter the antrum by removing first the external wall of the tympanic vault through the external auditory meatus. I have now done this and must expose a larger area of dura because a small dural exposure is always dangerous. I have now exposed a large area of dura and find that the dura has already been a large area of dura and find that the dura has already been

perforated by disease. I now form the radical cavity, taking down the spur as low as possible, using my horizontal semicircular canal as a guide. As you can see, I have now taken down the posterior wall of the canal so that the posterior tympanic space is completely obliterated. I have also taken down the floor of the canal so that the hypo-tympanic space is completely obliterated. Note that the bent probe introduced into the tympanic cavity can be drawn downward along the floor of the meatus without meeting any obstruction. Note also that the bent probe can be drawn from the tympanic cavity along the posterior wall without meeting any obstruction. I have also cleared out the Eustachian tube perfectly by means of the curet.

Dr. Dench then proceeded to demonstrate the method of grafting already described above. As a perforation had been found in the dura, the grafts were then removed, the dural opening enlarged by two crossed incisions, and the exposed brain area covered with iodoform gauze. The radical cavity was packed with iodoform gauze and an isolation packing was introduced between the packing over the exposed brain substance and the packing in the radical cavity. The posterior wound was closed and the patient returned to the ward.

The operator laid great stress upon the necessity in all cases where solution of continuity of the dura is found at the time of the radical operation, of the necessity of enlarging the opening and packing off the exposed area of brain with iodoform gauze. In this way the danger of meningitis becomes absolutely nil. A small opening in the dura is a dangerous condition. A large opening, properly treated, is seldom followed by any untoward result.

*Note.*—At the time of compiling these notes, the author wishes to say that the patient left the hospital, in perfect health, one week after the operation.

# TECHNIC OF THE EXAMINATION OF THE VESTIBULAR APPARATUS. A DEMONSTRATION.

By ISAAC H. JONES, M. D., Philadelphia, Pa.

Before this patient comes in I want to explain the situation. He is very intelligent and we do not want him to have the slightest suggestion that we consider him to be in a serious condition. He regards himself as perfectly well, and yet the ear-tests point strongly to a lesion of the brain-stem. First, I will ask Dr. Walter Roberts to give the history of the case.

Dr. Walter Roberts: This boy is a student at Swarthmore College. I was called to see him several weeks ago. The mastoid was tender but there was no swelling; there was, however, some swelling of the posterior superior wall of the meatus. Ten days before I saw him he had recovered from pneumonia, had gone out too soon and taken cold; then this trouble with the ear had started, soon developing a purulent discharge. Mastoid swelling appeared a few days later and I did an extensive Schwartze operation; the lateral sinus was exposed for quite a space—I think by the disease and not by the curetment. After operation he still continued to run a septic temperature. There was no evidence however of meningitis or sinus thrombosis. His recovery was slow and although the wound was healthy the septic temperature only gradually disappeared. Suddenly, just as I thought he was well, he developed dyplopia. At this point Dr. Jones was called in and we examined the boy together.

Dr. Jones: Examination; douching right ear showed absolutely no responses in nystagmus, vertigo, past-pointing or falling either from the horizontal canal or from the vertical canals. Douching left ear—the vertical canals gave no response, (douching with head upright). On tilting the head back however, there immediately appeared a normal nystagmus, vertigo and past-pointing. As the left horizontal canal and the left cochlea appeared normal, the absent responses from the left vertical canals suggested an involvement of the fibres from the left vertical canals in the pons.

The paralysis of the right external rectus has cleared up entirely, the boy feels perfectly well, is pronounced cured from a neurological

*Demonstration before the Eastern Section of the American Laryngological Rhinological and Otological Society; Jefferson Hospital, March 3rd, 1917.

standpoint, and would be allowed to go back to college and take up his regular work *if it were not for the ear-tests.* The ear-tests indicate that he is worse than he was at the time of the original examination. The additional features to indicate that he is worse are:

1. Right cochlea was functioning; Dr. Roberts noted that the Weber was referred to the right ear and that tests by air-conduction and bone-conduction showed that the right cochlea was uninvolved. The right cochlea is now absolutely dead.

2. The left horizontal canal originally produced a normal horizontal nystagmus to the right. The left horizontal canal now produces a "perverted nystagmus"—an oblique nystagmus, up and to the right.

(The patient was now brought in.)

*Examination.* Instead of asking the patient to look to the right and to the left, he was asked to fix his eyes on some object and the turning-chair was rotated first to the left and then to the right, thus causing the body, as it were, to be revolved around the eyes. (This simple procedure is especially useful in testing unintelligent patients.) No spontaneous nystagmus is observed.

Quite incidentally in this case we have the opportunity to demonstrate the classical statement that the endolymph current toward the ampulla is twice as strong as the current away from the ampulla. We know that in this patient the right end-organ has recently been destroyed. On turning the patient with the head 30 degrees forward we know we are testing the left horizontal semicircular canal. Turning to the right we should expect two-thirds of the normal nystagmus. $\frac{2}{3}$ of 24=16. Test done; stop-watch showed 15 seconds—just one second short of exact mathematical calculation. Turning the patient to the left, causing a current away from the ampulla in the left horizontal canal should produce one-third of the normal duration of nystagmus. $\frac{1}{3}$ of 24=8. Test done; stop-watch showed 7 seconds—just one second short of absolute mathematical perfection. Turning to the right gave 15 seconds and turning to the left gave 7 seconds; the current toward the ampulla of the left horizontal canal has produced twice as strong a response as the current away from the ampulla. This is merely incidental in this examination but is unusually clean-cut from a scientific standpoint.

Dr. Jones suggested it would perhaps be unnecessary to waste time in douching the right ear; on all examinations the right ear has failed to produce any responses either from the horizontal

canals or from the vertical canals. The left ear was douched, head 30 degrees forward—influencing exclusively the left vertical semicircular canals. No response whatever was observed—no nystagmus, vertigo, past-pointing or falling. The head was then tilted back 60 degrees in which position the caloric effect influences only the *horizontal* canal. Immediately a nystagmus occurred, but it was the *wrong kind of a nystagmus*—instead of being a horizontal nystagmus to the right which any normal individual would show, it was a pure vertical nystagmus upwards. This observation was corroborated by Dr. Eagleton of Newark who was asked to observe it closely. The patient's head was then tilted 120 degrees forward; any normal individual would then show a pure horizontal nystagmus to the left. There occurred instead a pure vertical nystagmus *downward*.

It is obvious we are dealing with a lesion of the brain-stem. No lesion of the right labyrinth or an epidural abscess in that vicinity could produce an absolute failure of responses from the left vertical canals, the fibres from which are evidently involved in the pons. This is corroborated by the perverted nystagmus on stimulation of the left horizontal canal; a perverted nystagmus after stimulation of any canal is pathognomonic of brain-stem involvement.

Dr. Jones asked advice as to the probable nature of this lesion. Is it an exudative or serous collection in the right cerebello-pontine angle subsequent to a basilar meningitis which had involved the right 6th nerve? Is it an abscess which should be drained?

This is the chart used in routine examination of the internal ear and its intracranial pathways.

Name Date

Address

Referred by Age

DIAGNOSIS

SUMMARY

Complains of

HISTORY

Dizziness
Staggering
Deafness
Tinnitus

NOSE

THROAT

EARS

A. D.
A. S.

Fistula

Hearing Tests

| A | A | c4 | Ac | Bc | n | Galt. | Pol. |
|---|---|---|---|---|---|---|---|
| = | | | Ac | Bc | n | | |

## TESTS OF THE VESTIBULAR APPARATUS.

### SPONTANEOUS

| NYSTAGMUS | | | POINTING RIGHT | POINTING LEFT |
|---|---|---|---|---|
| Looking to RIGHT | | Shoulder from above<br>Shoulder from below<br>Shoulder from side | | |
| Looking to LEFT | | | | |
| | Nystagmus<br>Vertigo<br>Past-pointing<br>Falling<br>Romberg<br>Turning head to right<br>Turning head to left<br>Attempt to overthrow<br>Goniometer | | | |

### TURNING

| | | | | |
|---|---|---|---|---|
| To RIGHT | | | | |
| Amp. | | To RIGHT | | |
| Duration | Sec. | Shoulder from above | to | to |
| | Nystagmus<br>Vertigo<br>Past-pointing<br>Falling | | | |
| To LEFT | | To LEFT | | |
| Amp. | | Shoulder from above | to | to |
| Duration | Sec. | | | |
| | Nystagmus<br>Vertigo<br>Past-pointing<br>Falling | | | |

### CALORIC

| | | | | |
|---|---|---|---|---|
| Douche RIGHT | | Douche RIGHT | | |
| Amp. | | Shoulder from above | to | to |
| After min | sec | | | |
| | Nystagmus<br>Vertigo<br>Past-pointing<br>Falling | | | |
| Head Back | | | to | to |
| Douche LEFT | | Douche LEFT | | |
| Amp. | | Shoulder from above | to | to |
| After min | sec | | | |
| | Nystagmus<br>Vertigo<br>Past-pointing<br>Falling | | | |
| Head Back | | | to | to |

DISCUSSION.

**Dr. Stauffer:** I think the left labyrinth did give reactions. The turning shows this.

**Dr. Jones:** The turning tested the left *horizontal* canal. The left vertical canals failed to react.

**Dr. Stauffer:** The vertical nystagmus shows that the vertical canals do work.

**Dr. Jones:** No, the vertical canals gave no responses whatever. No douching of any canal could produce a vertical nystagmus. If the left vertical canals had produced a response it would have been a rotary nystagmus to the right but the left verticals produced no response at all. The vertical nystagmus we saw was produced by the left *horizontal* canal which should normally have produced a pure horizontal nystagmus. The vertical nystagmus, therefore, is a "perverted" nystagmus.

**Dr. Mackenzie:** Did you try turning the patient with the head back?

**Dr. Jones:** Yes, there occurs a noticeably impaired nystagmus. In this connection I might speak of another case in which all other methods of examination failed to suggest an organic lesion. A middle aged vigorous man complained of headache; the ear-tests suggested a lesion in the IV. ventricle. The medical and neurological diagnosis was hysteria Death in two weeks—autopsy showed abscess in the IV. ventricle which in turn extended into the III. ventricle and the lateral ventricles, both of which were distended with pus; a sinus from the right lateral ventricle led to a large temporosphenoidal abscess which in turn communicated through a sinus to an old mastoid cavity on which a radical operation had been done 12 years before.

*Summarizing.* This boy shows definite involvement of the brain-stem but we are unable to say just what is the nature of the pathological lesion. One thing is sure—the ear-tests demonstrate that this boy is *far from well* although all other methods of examination indicate a perfect recovery.

**Dr. Clarence J. Keeler:** Dr. Jones has presented a case of intracranial lesion and as he has so exhaustively demonstrated the differential points of intracranial localization, I will show a patient with an end-organ lesion.

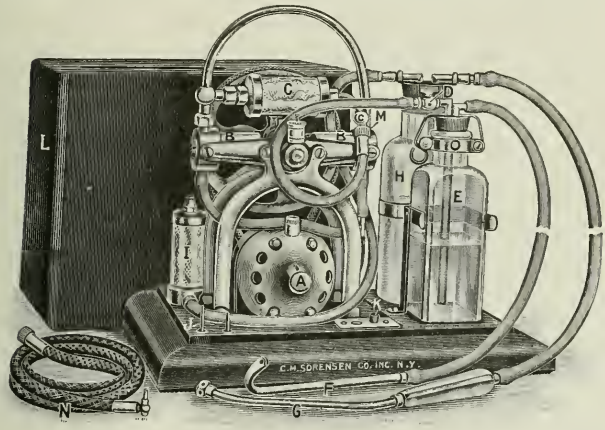
Man, aged 34, was thrown from a freight train and was unconscious for twenty-four hours. There is greatly impaired hearing in the left ear but Weber is referred to the left. Otoscopy shows a small hemorrhagic area in the drum membrane centrally located. Persistent roaring tinnitus; also vertigo and staggering. Turning showed a markedly exaggerated nystagmus, vertigo, past-pointing and falling. The caloric test also showed excessive reactions. It is evident we are dealing with an irritation of the labyrinth.

**Examination.** Turning shows a normal nystagmus and past-pointing. This corroborates the diagnosis and indicates that the irritability of the labyrinths has disappeared.

**Dr. Randall** called attention to the fair facility with which these tests were made, and the extreme importance of their being made with great accuracy, and recorded with precision in order that they may be compared with other cases for future reference. He deplored the habit of some men of endeavoring to carry the details and results of their examinations in their heads. He expressed his pride in the work done by what he called his group of "medical children" at the University, along the lines demonstrated by Drs. Jones and Keeler.

## DEMONSTRATION OF THE "VACUUM CLEANER" IN NASAL PRACTICE.

Dr. Haskin, New York City, gave a practical demonstration of the use of the Sorensen suction apparatus with a description of its various parts. He considered suction to be a practical, non-painful method for withdrawing secretions from the nose and sinuses. He considered it an immense improvement on the older methods of spraying and washing in catarrhal cases when the secretions are not easily cleaned out. By the use of the suction apparatus the nose




and nasal sinuses are quickly cleaned and the patient experiences prompt relief. In cases of peritonsillar abscess he found this apparatus of great assistance, placing a trocar in the tube and plunging it into the abscess and thus evacuating it. He felt that most of his success in curing chronic otitis media cases was due to his use of suction. He inserted the tube directly into the canal, drawing the secretion out of the eustachian tube.

## DEMONSTRATION OF ESOPHAGOSCOPIC BOUGINAGE.

Dr. Jackson, Pittsburgh, Pa., exhibited various specimens of foreign bodies removed from the bronchi of children. He described the case of an infant nine months, brought in to the Children's Hospital with a history of an attack of sudden choking and inability to swallow. He removed a large mass of gauze, a plumstone, a fragment of a button and a long roll of wool. He exhibited a case, a man twenty-one years of age, who complained of inability to swallow solids and liquids except with greatest difficulty. Upon examination an esophagitis was found, with a firm, tight constriction. The causation of this being vague, an abdominal examination was made and an abdominal malignancy was discovered. Specimens from both esophagus and intestine were carcinoma, indicating, as suggested by Dr. De Costa, an implantation metastasis. The age, 21 years, was very young for malignancy. A second case shown was that of a little child who, as a result of swallowing lye, had suffered from stricture of the esophagus. The stricture had been dilated, the esophagoscope having been used on three occasions previous to the demonstration. He stated that in adults almost any sized tube might be used, but in children the tube must be small. He then demonstrated the use of the esophageal bougie on the child, asking if it were not better, in such cases, thus to work without anesthesia rather than to have the child repeatedly etherized over a period of many months. He does not use even local anesthesia.

## EXHIBITION OF MENINGITIS CASE, POSTOPERATIVE.

By FRANCIS R. PACKARD, M. D., Philadelphia, Pa.

Dr. Packard stated that the patient, a girl of eleven years, had suffered an acute grippal attack, during which she developed an abscess of the right ear. There was vomiting and headache. The temperature was 102°, pulse 120. He incised her right ear drum, and the next day she showed mastoid and meningeal symptoms. She was admitted to the hospital and he did a mastoid. While she was under ether a lumbar puncture was done, giving turbid spinal fluid and the typical picture of acute meningitis. Noguchi's butyric acid test was positive; Fehling's copper solution was not reduced. Microscopic examination showed many pus cells, almost all being polynuclears. There were also some organisms resembling influenza bacilli. She received 30 c.c. of anti-influenza serum, repeated three times and the meningeal symptoms cleared up. She had had a positive Kernig's sign, but this also had cleared up. Convalescence was uneventful until October 29th, sixteen days after admission to the hospital. The mastoid wound had practically healed. She developed a slight temperature and complained of headache. There was no Kernig and no rigidity of the neck. Two days later she had several vomiting spells and examination of the eye-grounds showed the right optic disc to be hazy. The indications pointing to brain abscess the right temporosphenoidal lobe was opened and a large abscess evacuated. Dr. Packard thought the case interesting because the culture of pus from the ear showed pure staphylococcus, that from the brain abscess pure streptococcus, and that from the spinal fluid, bacillus influenzae.

## MEETING OF THE MIDDLE SECTION, HELD IN COLUMBUS, OHIO, FEBRUARY 26, 1917, UNDER THE CHAIRMANSHIP OF DR. JOHN E. BROWN, OF COLUMBUS.

### CASE REPORTS: FATAL CARDIOSPASM IN AN ADULT: LYE STRICTURE. WITH LANTERN SLIDES.

By THOMAS HUBBARD, M. D., Toledo, O.

The act of deglutition is initiated by the hypo-glossus and glosso-pharyngeus and finished by the vagus. Sensitive areas in the lower pharynx start the active, forceful contractions and esophageal peristalsis carries the bolus through the cardiac orifice. The lower esophagus has scant supply of sensory fibres and the muscular contraction is an active recovery from the distension. Sympathetic nerve fibres link the esophageal with the visceral nerves, the whole comprising an important part of the vegetative system. The act of emesis is a reversion of deglutition, the initial forceful impulse coming from below. No wonder that the esophagus, subject to frequent trauma and conflicting reflexus from above and below, gets "nervous" and manifests it by *globus hystericus* and *cosophagismus*. Emotional excitation, trauma incident to lumpy or too hot or cold food and active gastric disorder furnish the excitation for these more or less transient spasmodic disturbances. Cardiospasm is, as a rule, more serious and persistent. The inference is that the source of the disturbance is a more chronic lesion together with hypersensitive nerves; and since we find that the lower esophagus has intimate relations, not only with all of the pneumo-gastric tract but also with the abdominal viscera, we appreciate the wide range of perturbing reflexes. In other words we must study cardiospasm from the broad neurologic standpoint and at the same time take into consideration the probable pathologic lesions of a very considerable portion of the vegetative system.

Vagotonia and sympatheticotonia are the technical neurologic terms applied to subjects who exhibit unbalance of the vagus and sympathetic systems. Functional disturbances such as we are considering, and including asthma, are manifestations of vagotonia with more or less of the sympathetic factor. Clinically vagotoniacs and sympatheticotoniacs are not clearly defined but the terms are very suggestive and seem peculiarly applicable to the serious types of esophagismus and cardiospasm.

The case I am about to relate exemplifies many of the important points suggested in this cursory review of the nervous factor in

cardiospasm. The fact that such a disturbance is so resistant to treatment, so persistent in spite of well directed therapy impresses the fact of its entity. The two nervous factors in the vegetative system—the vagus and sympathetic—must be profoundly perturbed, either because of acquired or natural instability, to produce a degree of functional disharmony that may actually threaten life. One is reminded of those desperate incurable cases of bronchial asthma—a daily bronchismus, if I may use the term as comparative with that distinct type of esophagismus known as cardiospasm. To be sure there is a factor in common in both of these diseases conducive to chronicity. Emphysema and impaired elasticity of the lungs in the one and permanent dilatation of the esophagus are real pathological lesions and as such complicate and may even determine chronicity and prevent restoration of normal function. Food hunger and air hunger differ only in the degree of actual danger to life. But cardiospasm is not controlled by physiologic inhibition such as prevents asphyxiation in asthma.

We summarize the essential pathology of chronic cardiospasm as follows: An unstable vagus and sympathetic system susceptible to reflex irritation from a visceral lesion or abnormally responsive to emotional or psychic influence. Dilatation and food retention with consequent changes in musculature and mucosa may become factors in chronicity.

*Case report*: I. W., a large vigorous man of 58, actively engaged in farming up to ten years ago and since then retired. At the age of thirty he had heart symptoms, palpitation and subsequent general weakness lasting about three months. A mitral lesion was in evidence but compensation was gradually restored. Again at the age of forty-three he had prostration from which he recovered more slowly. About six years ago (aet. 52) he noticed discomfort in the lower mediastinum after eating, usually due to very hot or very cold effects and would often regurgitate the retained contents of the esophagus. Then he would eat heartily and relish his food. This would occur almost weekly but gave him no serious discomfort up to July 1915. At this time he regurgitated beefsteak twenty-four hours after eating. Dilatation was evidently well under way. On August 10th he regurgitated a dinner as swallowed and from that time to September 10th very little passed into the stomach. Even liquids were returned. A radiograph at this time showed an enormous dilation of the lower esophagus. For four days he retained nothing. Esophagoscopy was tried September 9th and the most accomplished was cleaning out of the enormously dilated esophagus—more than a pint of milk, curds and eggs with other detritus was removed by aspiration, swabbing and forceps. This took some time and as the patient was in extreme weakness from water hunger, the heart behaving very badly, I made only a few attempts to find the natural orifice. The mucosa was inflamed and edematous from the decomposed retained ingesta of a long period and it was evident that the esophagus now cleaned out should have time to recover from the inflamed state before attempting dilatation

of the stricture. At this time we couldn't exclude malignancy. Dr. Jackson was consulted by wire and reiterated what he states so forcefully in his book. *The water starved patient is a dangerous surgical subject.* I emphasize what he says because I am confident that continued effort to enter the stomach from above would have been fatal in this instance, partly because of the inflamed, swollen condition of the mucosa and partly because of the difficulty of subsequent feeding. In accordance with Jackson's advice gastrostomy was done under local anesthesia by Dr. J. H. Jacobson. The patient rallied and gradually gained in strength. He was fed satisfactorily for two weeks and then esophagoscopic search for the orifice was successful, the 18, 24, and then 36 Fr. bougie passing quite easily. From that time the cardiospasm was controlled by passage of a 36 or 40 bougie at intervals of a few days. The gastric incision gradually closed. But it became apparent that the old cardiac trouble was again active—daily fever, an occasional chill, with marked prostration were the chief symptoms. The endocarditis diagnosed by Doctors Stone and Grosh seemed to account for his general decline but throughout this period there persisted the characteristic spasms of esophagus, stomach and even intestines. The *cardiospasm* would recur if the bougie were neglected. Early in December he had two attacks of *spasmodic stricture of the colon* each lasting two days. *Spasmodic vomiting* persisted throughout even though there was no demonstrable gastric trouble. He would retain and digest food perfectly for a time and then would have days of spontaneous emesis without apparent cause. On December 16th he had a convulsion of an unusual type. Dr. Waggoner observed the case through four of these attacks. They began with rhythmic contractions of the diaphragm lasting several minutes, then the abdominal recti took part convulsing the whole trunk. The muscles of the neck were next involved. Actual restraint was necessary to keep him on the bed. He suffered great pain but remained fairly clear mentally; no positive evidence of a central lesion. Four of these attacks in three days produced a fatal exhaustion and he died December 20th.

*Autopsy*: The left lung was bound by pleuritic adhesions from an attack of pneumonia when young. The esophagus was dilated and the wall thinned the whole length, the maximum of fusiform dilation being at the middle third. The mucosa showed evidence of chronic inflammation but no ulceration. There were no signs of stricture fibrosis or muscular hypertrophy at the hiatus or cardia. The stomach was normal and the adhesions to the parietal wall very slight. The spleen was enlarged and had metastatic infarcts scattered over the cortex. The heart showed evidence of old inflammatory deposits on the valves and also fresh inflammatory vegetations. Active endocarditis as well as aortitis was in evidence. The kidneys showed slight pathologic changes. Liver normal. Brain not examined.

In this case we find two sources of vagus irritation. The endocardia including aorta, and the spleen. Such lesions as these are very commonly present with only the usual symptoms. The primary vagus explosions can be accounted for by assuming a progressive vagotonia and consequent local spasms followed by permanent lesions.

Certain it is that the irritable vagus, the enormously dilated esophagus, and the bad heart are the chief clinical and pathological features of this case and the clinical history emphasizes the importance of a broad, comprehensive study of cardiospasm. Treated in its incipiency, before dilation was established,

this disease could probably have been cured; but dilation having progressed, food retention having become a constant factor, the cardiospasm from whatever reflex irritation primarily, became chronic and ultimately the cause of death.

*II. Abstract case report. Lye stricture.* Boy age six swallowed lye February, 1911, gradual closure. In November 1911, impervious.

Sent in to hospital for gastrostomy. Well nigh starved—no fluids passed into stomach for four days. Emaciation and extreme exhaustion. On first esophagoscopic examination passed small whalebone bougies in group and then left a small catheter wired to teeth *in situ* for subsequent feeding. Removed in twenty-four hours and eggnog passed into stomach for a few days. Patient recuperated rapidly. Stricture begins lower third and is tortuous. Two or three inches of fibroid tube. Bougies were passed—up to 21 Fr.—once a week and patient did well up to March (five months). Then stricture closed again and I had considerable difficulty in passing even Sippey guide through esophagoscope. In fact had to use filiform whalebone bougies first. After that dilatation the passage was kept clear for a month. As it seemed inclined to close tried the string guide method. Good anchorage, in fact protruding at rectum in four days, and no trouble after that to pass Sippey guide wire threaded on string and then dilate with olive sounds in groups passed over the wire. Gradually dilated up to 31 Fr. and patient swallowed any kind of food. He became healthy and vigorous. Dilatation once in six weeks sufficed and it looked like a cure.

Treatments had covered a period of two years, probably forty dilatations in all. The boy died of acute septic meningitis—a four days' illness. No apparent relation to the esophageal trouble.

# CASE REPORTS OF FOREIGN BODIES IN THE BRONCHI AND ESOPHAGUS.

By JOHN W. MURPHY, M. D., Cincinnati, O.

I think that we can sometimes learn more from our failures than our successes, and with the hope of hearing what has been the experience of others, doing this kind of work, I thought it might be well to report some of the unpleasant experiences that have fallen to my lot with tube cases, lately.

I do not know how it affects the other men doing this class of work, but I must confess that I have spent more sleepless nights and restless days in worrying over my tube cases than all of my other professional troubles combined.

About the time that you are beginning to feel, after a series of successful extractions, that your equipment and experience are sufficient to encourage you to feel that you can approach the next case with the confidence and assurance that you are going to succeed, and this confidence is a wonderful asset in this line of work, when an apparently easy case comes along that proves your undoing. Then you wish the other fellow had gotten that case, since his results could not possibly have been worse and might have been better.

The more I see of this work the more I am convinced that every foreign body in the bronchi or the esophagus is dangerous, and the family physician, who generally comes with the case, the parents or friends should be informed that all of these cases are serious.

I am sure some of my failures were due to a failure to carefully consider all angles of the case, and to provide beforehand for any emergency that might suddenly arise during the attempted extraction.

Skilled assistants are absolutely essential, and without them even the most elaborate equipment may be of little avail.

I know of no work where time is such a factor in the case, and yet we should never be in too much of a hurry.

If the condition of the patient is not too alarming, a careful physical examination should be made, and time taken to study the case if the suspected foreign body is of such nature that its exact location can be determined by the X-ray, we should not fail to call to our aid the roentgenologist.

The patient's description of where the foreign body is located is not always reliable, as was well illustrated in the case of a fourteen-year-old boy, brought in from a neighboring town several weeks ago, with the history of having swallowed a pin, several days before. The patient could locate the position of the pin very definitely in the neck, just back of the thyroid cartilage. He had been taken to a surgeon with a view of having the obstruction removed by an operation. I saw the case in consultation, and insisted upon an X-ray examination before attempting an extraction. The pin was found quietly resting in the lower bowel.

In another case of a three-year-old child, suspected by the mother of having choked on a water melon seed, the X-ray disclosed a jackstone in the esophagus.

In several cases of a foreign body in the lower end of the esophagus, the only pain complained of was high up between the shoulder blades. Hence, the patient's feelings as to where the body is located, are not a trustworthy guide.

One of the most difficult problems for me to learn is when to quit. I am satisfied several of my bad results were due to too prolonged attempts at extraction. Once you have seen the foreign body, it is next to impossible to quit, even when the patient's condition warns you to stop. You feel that each attempt to grasp the body is going to be successful, and the examination is almost sure to be prolonged beyond the point of safety.

In bronchoscopic cases, the question of doing a tracheotomy is often a most perplexing problem to solve.

To many men, to do a tracheotomy when the patient is doing apparently well, is a confession of failure, yet it is a mistake to wait too long or until the patient's condition is almost hopeless. Just when this surgical proceedure should be resorted to has proven most perplexing to me.

I always have the instruments ready, and I am sure the prompt resort to this remedy has resulted in saving several cases.

*Anesthetics.*

When to resort to an anesthetic is often difficult of solution. Most bronchoscopic cases are poor anesthetic risks, especially if the breathing has been such as to weaken the patient's resisting powers too much.

In young children who can be easily held, I prefer to make my first attempt without an anesthetic, either local or general, and with the patient in the upright position.

Personally, I work much more rapidly with the patient in this position. This examination should not exceed ten or fifteen minutes, as the shock is generally quite pronounced. Should you not succeed in this length of time, it is better to allow the patient a few hours rest and then make another attempt under general anesthesia.

The three fatal cases I report today are all of my fatalities in one hundred and twelve consecutive cases, so my mortality is a fraction over two and six tenths per cent. (2.6%), but they have all occurred recently when I feel that my equipment and experience should have given better results.

So many perplexing problems are constantly arising in this line of work, and some of my results so discouraging, I thought a report of the following cases might at least show that foreign body cases are dangerous.

*Case No. 1.*

In November, 1914, a child six years old was brought to the hospital on account of very difficult breathing, with the following history: While riding with the father, who was hauling corn in from the field, the child was supposed to have bitten off the soft end of an ear of corn, and the same was inspired into the trachea. The child had been brought 25 miles in an automobile, and the foreign body had been in position about eight hours. The condition of the child from dyspnea was bad, and the emergency was such that an attempt at extraction was made at once. No anesthetic was used and the child was held on the doctor's lap, the family doctor having come with the case. No difficulty was experienced in introducing the bronchoscope, and the foreign body was seen, about the size of a small cork. Before it could be grasped, it was suddenly inspired deep into the bronchus. The child immediately collapsed and ceased to breathe. The instrument was withdrawn and artificial respiration administered. Getting no response, a rapid tracheotomy was done, but the child never gasped after the foreign body was inspired.

*Comment:*

I feel in this case that a mistake was made in attempting an extraction with unskilled assistance. I also under-estimated the gravity of the case. I reasoned, if the foreign body was in the trachea as I had every reason to believe it was from my examination, that its extraction would be a comparatively simple matter. I had not figured on what might occur and what did occur when the foreign body was deeply inspired with such disastrous results.

*Case No. 2.*

Child, twenty-one months old, was sent in from a neighboring city, April, 1916, with the history of having inspired a bean, ten days before while playing with a bean bag, which it had gotten open. The breathing became quite difficult at times, then the child would have some hours of comparatively comfortable breathing.

When I saw the case, the child showed the effects of prolonged difficulty in breathing, and the exhaustion of a five hours' ride in the train.

Physical examination showed the child was in a weakened condition from refusal to take food,—temperature 102°, pulse very rapid and breathing

difficult. Both lungs were so full of mucous rales that no foreign body could be localized.

As the child was fast wearing itself out, we decided to attempt an extraction at once. The breathing was so labored that no anesthetic was employed. Some considerable difficulty was experienced in introducing a tube of sufficient size to be of much use. The trachea seemed unusually small. So much mucus was encountered, that our progress was exceedingly slow, and I was never able to get a view below the bifurcation on account of the small tube used and the immense amount of mucus encountered.

The examination occupied forty-five minutes. No foreign body was found. The child was returned to its bed in fairly good shape. Five hours later the breathing became quite difficult, temperature rose to 103°, and heart became very rapid, and a tracheotomy was done.

In spite of stimulation and oxygen, the child died twelve hours after it came from the operating room.

*Comment*:

This case was a poor risk to start with. Pneumonia was present and the child was in very bad physical condition.

The examination should have been made under an anesthetic as there would have been less shock, and the examination could have been made more quickly. Also a tracheotomy should have been done sooner.

The examination was prolonged beyond the point of safety.

Fifteen minutes should have been the limit in a case of this gravity, while I employed forty-five. No postmortem was held, so we do not know definitely the cause of the obstruction.

*Case No. 3.*

Child, twenty-one months old, was sent in July, 1916 from a neighboring town with the history of having swallowed a nickel five days before. The parents and family physician had waited, thinking the foreign body would pass on into the stomach. The child had refused to take nourishment for several days, so was in a very weakened condition. An X-ray plate showed the nickel to be situated just back of the cricoid, and it looked as though extraction would be comparatively easy. No anesthetic was used. In attempting to grasp the foreign body, it was dislodged and passed on into the lower portion of the esophagus. Thinking it might have passed on into the stomach, no attempt was made to follow it deeper into the esophagus.

After resting a few hours, another X-ray was taken and the coin was located in the lower end of the esophagus. No further attempt was made at extraction, as the child's condition grew rapidly worse, and it died nine hours after the attempted extraction.

*Comment*:

I am at a loss to account for this death. The examination was not prolonged, and a foreign body of this character is not generally considered dangerous.

The weakened condition of the child from its refusal to take nourishment, together with the shock, induced by examination, contributed to the fatal termination.

As I said in the beginning, all foreign bodies in the trachea, bronchi, or esophagus are dangerous, and the longer they are allowed to remain, before an attempt at extraction is made, the less able is the patient to endure the shock incident to an examination of this character.

## REMOVAL OF AN UNUSUAL FOREIGN BODY FROM THE UPPER ESOPHAGUS, WITH REMARKS ON PRELIMINARY TRACHEOTOMY IN SUCH CASES.

By NORVAL H. PIERCE, M. D., Chicago, Ill.

As I am an unskilled operator in this field, I take great pleasure in passing around the largest foreign body that I am acquainted with, namely, a bed spring taken from the esophagus. The man was suffering from dementia praecox, a resident of the insane asylum; thirty odd years of age. He had a long thin neck. Like many of us, he had an inordinate desire to do things that he should not, his particular temptation being to throw objects through panes of glass. He held this desire in check as a rule, but on this particular occasion he yielded to it, and threw a piece of iron through a priest's window at the asylum, and was punished by incarceration. While so incarcerated, in order to avenge himself against the world he proceeded to eat up his bed spring. This spring had been in his esophagus about six days when I saw him. Attempts had been made to extract it, but for some reason or other had been unsuccessful. When I saw him there was considerable edema around the aryepiglotic folds and the interarytenoid region. While he could breathe comfortably when not exerting himself, the moment he did exert himself there was a good deal of stridor, and if you touched this body it would produce a spasm of the glottis. The upper portion of the hook was visible above the arytenoids by simply pressing down the tongue firmly with a tongue depressor. The question arose as to whether we could get this foreign body out without an anesthetic, and without tracheotomy. He was very much opposed to having anything done. He was quite convinced that if this body was not disturbed it would "go down all right." He was led to that conclusion by the fact that eight nails had already gone down before, and he did not see any reason why this should not pass, so that he would fight if any attempt was made at extraction. Therefore, it was evident that we would have to give him an anesthetic. Now, I was fearful of what might happen during forcible anesthesia, and so concluded that tracheotomy would be advisable. To this

he did not object so tracheotomy was done under local anesthesia very easily. He was quite docile. After the tracheotomy was performed we anesthetized him with chloroform through the tracheal tube, and then extraction of the foreign body was very simple. Curiously enough it resisted considerable force, pulling directly, so that I unscrewed it. I gave it about two turns, and then it came out very easily. My explanation of that was that the edema, the infiltration of the tissue, had settled in around this spring, and that in unscrewing it we unscrewed it from this edematous infiltration of the mucous membrane. Everything went very well, and he recovered very rapidly.

It is very unusual that we should be called upon to do a tracheotomy in esophageal foreign bodies. However, it sometimes becomes necessary, as I know of a couple of other cases where the foreign body, situated in the upper portion of the esophagus, has resided there for sometime, producing edema of the epiglottis and arytenoid bodies, and in all such cases I believe it is best to perform a tracheotomy before going ahead with the extraction of the foreign body. This, of course, is true of foreign bodies in the upper portion, in cases like this, where resistance to extraction is sure to occur. In other words, I am inclined to always do a tracheotomy when I have the least suspicion that it is going to be of any value whatever. This is because, as I said at the beginning, I am rather an unskilled operator in the removal of foreign bodies from the trachea, bronchi, or esophagus.

I think that the endeavor to escape tracheotomy has been accentuated too deeply by writers on this subject—by Jackson, for instance. I mean that it is better for the rank and file of men who are not so skilled, so wonderfully dexterous, not to hesitate to do a tracheotomy if it is in any way going to facilitate the operation. I believe with Dr. Murphy, that we should resort most readily to the administration of a general anesthetic. My impression is that my work is better carried on under general anesthesia than under local anesthesia in the majority of cases.

## DISCUSSION.

**Dr. J. C. Beck**, Chicago, Ill.: Mr. Chairman, Gentlemen—I do not see why the Chairman selected me to open this discussion, except that he knows that I know very little about this work. I rise to emphasize the point made by Dr. Murphy in reference to assistants. It has been my practice for six or seven years to always employ an expert in every case of foreign body in the bronchi that has come to me. I ask him to assist me, to the extent that he removes the foreign body, while I stand by and perhaps encourage him to quit or keep it up, just as the

case may be. I do not limit myself to one man. We have two or three men who do this work, and I ask all of these men. Dr. Friedberg has assisted me most, and has been most successful in saving the lives of a number of little patients. I refer all the babies to him, when it is a matter of dexterity in handling the instruments, and an important matter as to time. I can recall at least a dozen cases now, which I feel in my own mind would have gone very badly had I attempted the work myself. That is the point in reference to Dr. Murphy's case.

As to Dr. Hubbard's case, it is an interesting matter. I have had some experience in cardiospasm. I had a case which traveled from El Paso to Chicago to be relieved of this sort of condition which the doctor had mentioned. There was the artificial stomach, the dilated esophagus. We washed it with lavage, and subsequently used a cocoa butter suppository and then there was no difficulty in passing a sound; there was absolutely nothing organic in that case that we could discover. The man had hyperthyroidism, and it was subsequently proven that he had tuberculosis of the adrenal. The condition of the autonomic nervous system as an etiological factor in gastric spasms, intestinal spasms, anal spasms, and all conditions of spasms have to be considered. The disturbance of the autonomic nervous system is due to a disturbance of the glandular system of which we know so little definitely; yet it is proven in the laboratory, and also clinically, the influence that either a deficiency or excess of one or more of these glands has to do with this condition. I simply mention that this man had hyperthyroidism. We have no means of determining these factors chemically, except by feeding the patient a sort of gunshot prescription of various glands, until we finally find what it is that this patient must get, and then that particular gland therapy is pushed.

As to globus hystericus, a condition of spasm, I am surprised that the essayist did not mention fissures of the upper portion of the esophagus, which have been shown to be responsible in many cases. We find that there are fissures in the upper portion of the esophagus, and by stretching them, and touching with nitrate of silver, the globus hystericus—the pest of the clinic, is relieved.

In reference to Dr. Pierce's case, I think I have him beat on that, although I did not extract any of the foreign bodies myself. But my brother, Dr. Carl Beck, extracted some thirty pieces in which there were nine actual full sized knives in the stomach. Had I known the nature of this meeting I would have brought you the photographs in that case. That patient was also insane, and was brought to the Cook County Hospital under my service because a foreign body had gotten lodged—a very important one, a silver dollar. This man's esophagus was of enormous size when a silver dollar would pass. You would hardly believe that such a thing would happen, but that is exactly what took place.

Question: How far down did it go?

**Dr. Beck:** It went as far as the lower part, where the Doctor showed the stricture close to the diaphragm. These foreign bodies were removed. I would like to have Dr. Pierce in closing state whether intubation is not possible in some cases. I have had some experience with edema of the esophagus. A case was brought to me after an attempt had been made to remove a chunk of meat from the upper part of the esophagus. That was removed, but there resulted much edema which caused great embarrassment in breathing, and an intubation tube was inserted until the trouble ceased.

**Dr. T. J. Harris**, New York: I want to emphasize everything that Dr. Murphy has said in his paper as to the responsibility that we who do this work, little or much, feel and ought to feel in the care of our cases, and in fact the whole problem that confronts us. Strictly speak-

ing, I do not know of any department of Oto-Laryngology that requires more thought, and gives more concern than a tube case, so much so that if I can conscientiously get out of it I am glad to do so. I feel that the responsibility reaches far beyond the time when we have successfully extracted the foreign body, and done what is necessary to relieve our patient. One of the most valuable papers, if not the most valuable paper touching on this point, was that presented by Dr. Jackson at the meeting of the American Medical Association last year. He told us how essential and important it is to study our cases, and appreciate the problem before us, and to endeavor in the extraction, to do it so that we will not encounter the troubles so apt to follow forcible extraction.

I wish to report a case in a very few words.

My patient (a fatal case) was a man of 35 years. He came into the hospital with the history that he had in mistake at night, while gargling his throat, used instead of peroxide, ammonia. He recognized the error and was quite positive that he got it out before swallowing any. I saw him perhaps four weeks after the accident. At that time he was getting fluids down with difficulty. He was somewhat emaciated. No X-ray pictures taken before he entered the hospital. The esophagoscope was passed, and we encountered a stricture. This was quite in line with what has been said by Dr. Murphy. The stricture was just below the cricoid. The tube was passed into the stomach without difficulty, and he was immediately put under systematic dilatation. He came to my office during the months of July and August twice a week, and I was able to dilate that stricture up to 40 French. He was taking solid food. I left on my vacation the first of September, and instructed him how to pass the bougie, and he was passing his bougies without difficulty when I left. When I returned in October he reported to me immediately that he was in bad shape, and stated that he could only get down fluids again with difficulty. He was very weak, and very much emaciated. It seemed very clearly a case for the hospital and he was again sent in, and this time a picture taken with bismuth. We found the sac, spoken of here today, and the ring 28 centimeters from the mouth. Under cocain, (not under general anesthesia) we were able with a great deal of difficulty to find the esophageal opening, and introduce the closed end of a Jackson dilator, and then I dilated this stricture up to the full width that the dilator permits. The patient was sent back to the ward. Immediately he was considered a surgical case for a gastrostomy, and after consultation with the general surgeon we were preparing to do it, when the nurse informed me that the patient was able to take fluids. Within twelve hours after the dilating he was able to swallow fluids, and he kept on doing it. The interesting, or rather the sad part of this case followed, along the line of Dr. Thompson's report. Perhaps a week before the accident occurred the esophagoscope was passed. Having determined, as we thought, the position, we found no difficulty in passing bougies. The bougies were passed by one of my assistants daily for a week, and then as he was doing very well he came into the clinics, being sent down from the ward. I began again with a 28, and ran up to a 40 French, when I found resistance. There had always been resistance in passing the bougie. There was that firm wall which would always give, and then the bougie would pass on into the stomach. On finding the resistance that day I used a trifle more force than before. The man complained at that moment of some pain, not marked or severe. The next day the hospital called up and said he wanted to go home because his wife was very ill. The report was that he had had a good deal of pain over the lower part of the chest, but the house surgeon felt it safe to let him go home. Later I learned indirectly that he became very ill, and passed away at another hospital in a few days. I am sorry that I cannot give that as a fact, but I am sure it is the correct history. I have

no doubt that the same thing occurred in that case as occurred in the case which Dr. Thompson mentioned. The fibres of the sac had been dilated, there was no resisting power, and acute inflammation occurred, with death. The interesting point of my case was that the man undoubtedly had an organic stricture behind the cricoid which we dilated, and dilated successfully. What took place below has always seemed to me was a stricture of a functional nature, in other words, a so-called cardiospasm, dependent, of course on the condition of the upper end of the esophagus.

I cannot feel that those cases can be regarded with too great gravity. This case was a chronic case from June until October, then there was recurrence so that the patient was able to eat with difficulty solid food. I am thoroughly satisfied that while I did feel very much the gravity of this case, if I had used my esophagoscope, instead of depending on using the bougie by touch, my patient might have been alive today. Dr. Mosher brought out in a paper presented years ago in Boston. In fact all esophageal work should be approached with a great deal of circumspection. Dr. Jackson says that the esophagus tolerates abuse far less than the trachea or bronchial tubes. You can do much more in the way of harming the trachea or bronchial tubes than the esophagus. A wound of the esophagus at all, especially in children, is very apt to be followed by a fatal result.

**Dr. J. A. Thompson**, Cincinnati, Ohio: I would like to mention one point in connection with Dr. Hubbard's paper, which I have never seen in print, and I do not think it has made much impression on Dr. Hubbard, and that is, he does not note in his paper the dangerous thinning of the esophagus in these cases of cardiospasm. Two years ago one of the surgeons of Cincinnati referred a case to me for examination to see whether or not there was an organic stricture. The patient had had difficulty in swallowing for ten years. I passed the esophagoscope into a large sac such as we found in these cases. I could see the opening into the stomach, as the sac was clear, and there was no difficulty in passing into the stomach. After withdrawing the esophagoscope the doctor suggested that I pass a bougie, which I did with perfect ease. The patient swallowed for a week after that without trouble, and then returned to the surgeon with the old difficulty. He passed a full sized bougie, as I had done before. The patient immediately began to complain of extreme pain, in a few hours had a temperature of 104 degrees, and was dead in about 76 hours from mediastinal abscess. At the postmortem we got that esophagus, and in the lower 3½ or 4 inches of it the muscular fibres were exceedingly hard to find. The esophagus was mucous membrane, and serous membrane, with no muscular structure. In all these cases of chronic cardiospasm we are liable to find that condition. There is always danger in these cases if we attempt to pass a bougie.

**Dr. W. B. Chamberlin**, Cleveland: In connection with the case of stricture of the esophagus I would like to mention two cases. But before reporting these I want to call especial attention to the use of the Mosher scope. I have had experience with all other scopes, and there is no doubt in my mind of the increased freedom which this scope gives you. It has an elliptical, instead of the usual round opening, giving a wider lateral view. In connection with these two cases I also wish to mention the new Jackson dilator. I got my idea from Dr. Dean while recently visiting in Iowa City, and also from Dr. Friedberg, of Chicago. There are two prongs between which a disc comes down. This is controlled by the handle. One can insert this into a very small stricture, and then run the disc downward, separating the blades. Possibly some of you have had difficulty in passing even a small bougie through a stricture. After you have once gotten the first contraction

of a stricture broken by this dilator, it is comparatively easy to pass a bougie. In the two cases in which I have had occasion to use it I have found it especially valuable.

One case, a baby two years old, came into the hospital greatly emaciated, not only from food hunger, but from water hunger. A gastrostomy was at once performed, and the baby gotten into suitable condition for operation. We found a stricture of the esophagus, complete, about eight inches from the teeth. After considerable difficulty I was able to pass a very fine filiform bougie through the stricture. The openings were multiple, possibly three or four, or five. I located the most likely one, and passed a bougie down into the stomach, being very solicitous even with this fine bougie lest I had made a false passage. I thought it was going to be a comparatively easy thing to find the end of this bougie and pull it out through the abdominal opening; but I hunted for it for a long time. I finally found the bougie with a Kelly cystoscope, using an ordinary head mirror, and pulled it out through the belly wall. It was now a comparatively easy thing to run a string down through the stricture, and tie the ends together outside the body. I did not do anything further at that time, but merely left the string in place. A few days later I was much chagrined to find that the youngster had chewed the string in two. I then passed two strings, a large and a small one, and put on a very fine piece of rubber tubing to prevent the child again biting the string in two. The rubber tube irritated the mouth so much that we had to take it off and leave the string in place. Soon after this, from the dilation caused by the string, the child was able to swallow. The nurse, who was very resourceful, tempted the child with pieces of candy. Before that if even a glass of water was brought to the child it would yell and scream. I left the strings in position and did nothing else, relying on the dilation which they would afford; just as in the old days we used to pass a filiform bougie into the urethra and leave it in for the purpose of dilatation. Later I was able to get through this stricture with the Jackson dilator. We finally passed larger bougies, so that the last one passed was a 25 or 26 French. The abdominal wound is closing, and the child is eating practically everything.

Another very interesting case was that of a boy eight or ten years of age. He gave a history of having swallowed a toy pipe five years before. The interesting feature was that this pipe was in position for some five weeks, and although the child was seen by a very good man the diagnosis was made of a foreign body not in the esophagus but in the trachea; the reason being that the child could swallow perfectly. He had great difficulty in breathing, so much so that in the short period of five weeks he had developed a very typical and marked pigeon breast. The child coughed the foreign body out five weeks after swallowing it. The pipe as described (I did not see it) was composed of candy with a metal envelop, and this metal envelop had holes in it so that the food would pass through. A few months ago the child developed difficulty in swallowing. He could swallow soft foods and liquids, but not solids. At the examination we found the larynx and trachea absolutely normal. Immediately behind the cricoid we found a firm stricture, which we could not get through with any sort of bougie. We later tried to examine by means of the X-ray, but this was unsuccessful. The fluoroscope was then tried, and although I did not see the examination, it was reported that the bolus of food would fly down so fast that one could hardly locate it. We then made a second attempt to pass the bougie but failed. I now had the boy swallow a string before the next examination. In this case there were apparently several openings. On making the examination with the string in position, sliding the Mosher scope over the string, I found much to my surprise that the string went down what was to me the least likely of the openings leading to the stomach. I had tried all the others. I passed a bougie down along the string, but in pulling

out the bougie the string came out. I was afraid to do anything further, but sent the child to the ward, and had him swallow another string before the next attempt. At this attempt instead of using the bougie first I used the Jackson dilator, and dilated the stricture. I was then able to pass down a larger bougie without removal of the string, and was ultimately able to pass a small sized Mosher scope through the stricture. This stricture was one half inch in extent. The rest of the esophagus was absolutely normal.

In the first case why lye should pass down the esophagus and cause no lesion until a distance of eight inches from the teeth, is more than I can say. There was no scar formation in the esophagus or on the pharyngeal wall.

A word with regard to Dr. Murphy's paper and the upright position. In the early days we got in the habit of using the upright position. That was the position in which we did all our work, and we have found it difficult to change. I found it so myself. Now I use only the horizontal position, except in cases of adults. I am sure it is very much better. Another argument in favor of the horizontal position, is that if in the upright position the foreign body becomes dislodged gravity takes it down always. That is a very potent argument in favor of the horizontal position.

Speaking of anesthesia, I know that I have some very good men, certainly much better men and men of greater experience than myself against me; but in children, in spite of the well formed opinion to the contrary, I always use anesthesia, and I firmly believe that the shock of the operation with an anesthetic is very much less than the shock of trying to hold a struggling child. It is not only a shock to the child, but also a shock to the operator. It is not easy to operate when a patient is struggling. Personally I do not put myself in a class with Jackson. I believe he is in a class by himself—but the fact that he can do an operation without anesthesia is no argument to me that I can do it. He has had more experience than any man in this country, or possibly in the world.

**Dr. H. W. Loeb**, St. Louis, Mo.: Much has been said in the discussions, and in the papers in regard to inexpertness. What I want to say is that inexpertness comes from lack of cases that come to the individual men who are practicing laryngology. Secondly: to the lack of organization, which is of course an important feature. Thirdly, and not least by any means, to the circumstance that the instruments are seldom in order. There is a little expedient which we have adopted, and which I think can be recommended for smaller communities, as it has been of some service. For instance, Dr. Sauer and I try to see each other's cases and work together. I think a little work in this direction helps the individual man very much, and makes it possible, as it were, to assure some sort of organization, depending, of course, on the activity of the men doing this work. There are good rhinologists and laryngologists in every community, and if two or three men will work together in this sort of thing, I am sure that more than one case in a period of time will come to successful issue. I am willing to confess that in my own case I have not been as active in prosecuting this proposition as I should be, but I feel that results can be made much better.

I have listened to this discussion on cardiospasm, etc., and am rather surprised that so little has been said about the internist's side of this question. We all hear a great deal of the surgeon, the laryngologist, etc., but in my humble opinion if the internist is ever needed he is needed in these cases of esophageal stricture. I feel that that is so important that it should be stated in this discussion, that great help can be secured by the continual observation of the patient by an internist, in connection with this work.

**Dr. L. W. Dean**, Iowa City, Iowa: In our service at this time we have a child about 12 years of age with a perfectly normal pharnyx and esophagus down to the cardia, and there we have found a marked fibrosis of the membrane with the usual cicatricial stricture. My explanation for this case as well as the one mentioned by Dr. Chamberlin is that the child swallowed lye which was well diluted. It passed down the esophagus rapidly, and when it was checked at the cardia it produced an irritation which resulted in a temporary cardia spasm which retained the fluid until the caustic action took place, resulting in the stricture.

Dr. Hubbard's report of a case of multiple stricture reminds me of a case which we have now in our service which we have only relieved with great difficulty. Three months before the child came under our care she had swallowed some lye which resulted in an apparently complete closure of the esophagus. No water had been swallowed for at least two months.

A gastrostomy had been performed several months before the patient came to our hospital. On passing the tube we found a very small stricture near the cardiac end of the esophagus about one and one-half inches from the stomach. On passing a small bougie through this stricture it came in contact with what was apparently a solid fibrous wall. The second obstruction was not more than an inch below the first. I was unable to tell whether the second wall was complete or whether there was a second stricture eccentric to the first. I used a Jackson dilator and readily dilated the upper stricture, allowing me to pass a very small bougie and find a second stricture which was eccentric to the first. The case is now being dilated in the ordinary way, and the result so far is just fairly good.

I am very much interested in Dr. Hubbard's report of a case of cardiospasm, because for several years I have been searching in our service in Internal Medicine and in the Laryngological service for a case of cardiospasm and as yet have not had a single case diagnosed. Personally I have come to the conclusion that cardiospasm is exceedingly rare.

I would like to have Dr. Hubbard in answering, and Dr. Pierce, and Dr. Murphy, state their opinions as to the rarity of this condition which I have not seen.

**Dr. Murphy** (closing the discussion): Mr. President, answering these questions backward, I would say to Dr. Dean, as to cardiospasm, that I have also been watching for a case of this kind. I have heard and read a good deal, but have not yet found a case of pure cardiospasm. I had a case about a year ago in which I was ready to make the diagnosis of cardiospasm in a woman 62 years old, but examination with the esophagoscope showed that she had a very firm growth at the lower end of the esophagus. At that examination I dilated the stricture and for a week or ten days she did very nicely, and then it closed again. We then made an opening into the stomach, and I passed a tube of radium down through the scope. This stricture was so tight that it required considerable force to get through. The radium was allowed to remain over night. It was withdrawn the next morning, and then of course the woman was fed through the stomach opening. Whether it was the radium or the rest that helped that stricture, I am not able to say, but in two weeks time the woman left the hospital and was able to swallow perfectly. After several weeks she wrote me that she was so well, the only thing that annoyed her was the opening into the stomach, and she wanted it closed. I wrote her that if she would come to the city I would close the opening, but she shortly after took a cold and died.

Dr. Beck: How much radium did you use?

Dr. Murphy: Fifty milligrams.

Dr. Harris: What was your idea in using the radium?

Dr. Murphy: Because of the malignant growth present. Whether it was the radium or the rest that helped her I cannot say.

I have not really seen a case of cardiospasm.

Speaking of sword-swallowing, we had a rather amusing case when I was with Koehler. One day a professional sword-swallower came to the clinic, complaining that he had swallowed a bone which was sticking in his esophagus. On learning that he was a professional sword-swallower Koehler remarked that he didn't think a little bone ought to disturb a man of that kind.

As to Dr. Chamberlin's remarks in reference to position—that is a hard problem to solve. When you find the surest way to operate you feel like adopting that way, and the upright position is much easier for me than the recumbent.

However, I am coming to believe more and more that an anesthetic is the safer method than to try to hold a child. In my case in which the child inspired the nub of corn-cob, I believe that an anesthetic would have prevented struggling, and it was the struggling which caused the foreign body to be drawn deeper into the bronchi, with fatal result.

As to the case of stricture in which Dr. Iglauer was called—I see many cases with him. In fact, whenever I can I try to have as Dr. Loeb says, the assistance of someone who is competent to go ahead and see the case from different angles. He may suggest something which you do not see. These cases come in under great excitement, the family doctor is excited, everybody is excited, and the excitement is likely to be contagious. You feel that you have to hurry up and do something —get busy, when it is very seldom that that is the case. Dr. Iglauer had a boy from the mountains of West Virginia, with a typhoid stricture. He had an opening in the stomach, and I worked from the stomach end while Iglauer worked from the mouth. We brought our tubes together until the light could be seen between these and the thin stricture of the esophagus. Then Dr. Iglauer with that as a guide burnt a hole through at this thin membrane with the electric cautery point, and then passed in a bougie. I was watching for the lower end of the bougie, but I could not discover where the bougie was. I finally passed my finger into the stomach through the opening and found the bougie had come down the wall of the esophagus along into the wall of the stomach, so that a large part of the bougie had passed in and curled around the stomach wall. Another peculiar thing about this case was—the father insisted on taking the child home two or three days later. Half way home he wired back that the child had been able to eat a good meal on the train, and had been able to swallow very comfortably. What had happened to allow the child to do that we do not know, but it only goes to show that peculiar things occur in some cases. The child died several weeks later, but we do not know from what 'cause; possibly from a mediastinal infection, but we do not know. Why the child should have been able to swallow a day or two later was one of the questions we could not solve.

**Dr. Pierce** (closing the discussion): I have very little to add, except in reply to Dr. Beck's query as to whether intubation might not be the best means of overcoming the laryngeal stenosis in these cases. I should say that if it would be found that intubation was sufficient, we should perform intubation; but in all probability, in these cases where the edema is high up, the tracheotomy tube would be more advisable, inasmuch as the edema might mount over the laryngeal tube.

In my experience these spasms of the esophagus are exceedingly rare. I do not know that I have seen one for six years, and then the case I saw at that time was not in my practice. I think these cases go to the general practitioner more than to the specialist.

**Dr. L. W. Dean**, Iowa City, Iowa: Just a word, Mr. Chairman. We have a very large service of neurology, internal medicine and pediatrics in the University Hospital. In these three services any patient with a suspicion of an esophageal lesion is examined fluoroscopically, by means of the X-ray, and is referred to me for esophagoscopy. The heads of these departments as well as myself have been looking for a typical case of cardiospasm for a number of years, and up to the present time we have not found one case.

**Dr. W. B. Chamberlin**, Cleveland, Ohio: May I have another word, Mr. Chairman, in regard to position, apropos of Dr. Murphy's reply? I realize that my first dissatisfaction in regard to the position of a patient in the horizontal was due to the fact that I did not have a properly constructed table. I got the idea from Skillern of how important it was to have a table adjusted to the fraction of a degree. I was not able to get a Killian table, but we secured a table made in Cincinnati by the Max Wocher and Sons Company. It is on the same principle practically as the Killian, and is very efficient. I have used it in the neighborhood of a year. It is fairly inexpensive, costing some seventy or eighty dollars. To me it is extremely important to have a comfortable position while operating. If you have a stool adjusted to a nicety, and a table so adjusted that when you sit on the stool the heighth of the tube comes on a level with the eye, it is exceedingly valuable and important.

**Dr. Hubbard** (closing the discussion): Mr. Chairman, in reference to the discussion of Dr. Beck, I will say that I simply tried to emphasize the point that each case of cardiospasm should be approached from a neurological standpoint. In fact most of these cases ultimately find their way into the hands of an expert neurologist.

Pure cardiospasm with attenuation of the esophagus, was mentioned by Dr. Thompson. We should bear this condition constantly in mind. A thin walled esophagus with saculation should be treated with great care in the passage of bougies. The same applies to the use of the esophagoscope. A rupture is very liable to happen.

I have no doubt also that organic stricture in the upper portion of the esophagus is associated with spasmodic strictures below, and what passes for multiple strictures are in part spasmodic. It is almost impossible to eliminate the spasmodic element in all organic strictures.

The oval tube suggests a little history. The first tube passed into the esophagus for diagnostic purposes was passed by Kussmahl about 1840. He had a case of carcinoma of the esophagus. One evening he happened to attend a circus, and saw a man swallow a sword, and it entered his mind that if a man could straighten out his pharyngeal and esophageal tract to permit him to swallow a sword, he could pass an oval tube. He had it constructed and with a battery of lamps was able to actually study the carcinoma through the same type of tube that Mosher is using. That gave Killian the idea of direct esophagoscopy.

Dr. Dean's explanation of the location of the most serious erosion at the cardia in lye cases is probably correct. That is the point where the lye is temporarily checked in passing, producing irritation with slight spasm, retention and deep erosion. Of course in most of these cases we get slight erosion all the way down.

The care of instruments is a very important matter. If you go into a case with instruments working poorly you are anxious about them, and you cannot do your best work. In the care of tubular forceps, in fact all instruments of tubular character, or instruments of complex mechanism of any type whatsoever, if you soak them in spiritus saponatus, Ger. Ph., you will have little or no trouble with your instruments. This has to be made very carefully. If you will follow the German Pharmacopoea implicitly you will get a clear amber tinted liquid. It contains an excess of potassium hydrate sufficient to

sterilize any instrument. You need not be bothered with boiling to sterilize. You can take them directly from the solution, wipe them in sterile gauze and feel that they are sterile and also lubricated. It further makes sterilization with alcohol possible because the oleate is soluble in alcohol. If you put instruments in mineral oil or gasoline you cannot sterilize with alcohol, because alcohol does not dissolve mineral oil.

# I. TWO UNUSUAL BRONCHOSCOPIC CASES WITH SPECIAL INSTRUMENTS USED IN EXTRACTION.

# II. SPONTANEOUS EXPULSION OF AN UPHOLSTERY TACK FROM THE RIGHT BRONCHUS.

By SAMUEL IGLAUER, M. D., Cincinnati, O.

The following bronchoscopic cases present some unusual features both in the nature of the foreign bodies and in the methods of their extraction.

*Case I. Steel-Jacketed Bullet in the Left Bronchus.*

During the mobilization of troops in the summer of 1916, Edward R., aged six years, was given a U. S. rifle cartridge for a toy. The steel-jaeketed bullet came out of its shell and on July 7, 1916 while playing with the bullet in his mouth it slipped down his throat.

On the advice of his family physician, he was brought to the Cincinnati General Hospital three days later.

*Physical Examination* showed increased expansion and hyper-resonance over the right chest. The left chest showed impaired expansion and diminished resonance with breath sounds absent except in the apex region. The heart was transposed to the left with the apex beat in the fifth interspace 2.5 cm. from the nipple line.

A roentgenogram showed the bullet point downward in the left bronchus. The left lung gave a very dense shadow and the heart was transposed to the left. (Fig. I.)

*Operation.* July 10, 1916, morphia gr. ⅛ and atropine gr. 1/200 were given, and ether followed by chloroform was administered. An 8.5 cm. Kahler bronchoscope was introduced through the mouth and the foreign body was found firmly impacted in the left bronchus. With the ordinary forceps which I had available I was unable to grasp the foreign body so that I finally employed a flexible forceps, (Fig. II.) which I had designed for another purpose. Aided largely by touch imparted through this flexible forceps, the bullet was grasped by the blunt end and withdrawn with the bronchoscope. It was 25 mm. long and 7 mm. wide. (Fig. V, A.)

After operation the patient was slightly hoarse and showed evidence of a severe bronchitis but within a week was discharged in good condition.

*Case II. Umbrella Ferrule in the Right Bronchus.*

Carlos H., aged 12 years, was admitted to the Cincinnati General Hospital on January 4th, 1917. He stated that on the afternoon of the same day, while standing on the edge of the curb-stone with what he thought at that time was a small brass bell between his teeth, his small brother came up behind him and gave him a push and threw him off the curb. As he left the curb he gave a gulp and "swallowed" the bell. He at once had pain in his right chest and he had difficulty in breathing. He was rushed to the Cincinnati

General Hospital, though at this time he suffered very little pain except when he took a deep breath.

*Physical Examination.* The patient was at once taken to the X-ray room and the fluoroscope showed a thimble-shaped object in the right bronchus about two inches below the bifurcation. (The child was also given a glass of bismuth mixture to drink and the fluid could be seen passing down behind the object.) An X-ray plate was made at the same time showing the foreign body well down in the right bronchus (Fig. IV.) The respiratory expansion over the right side was not as marked as over the left. On percussion no

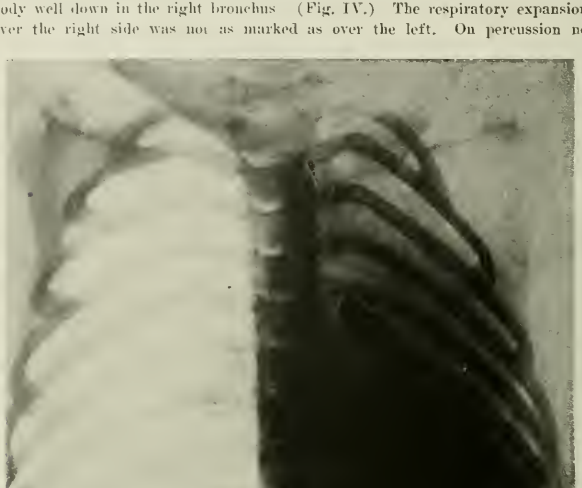


Fig. 1.—Roentgenogram showing bullet in left bronchus.

areas of dullness could be found. Auscultation revealed breath sounds in both lungs, but more feeble in the right, which lead to the conclusion that the foreign body either had a hole in it, or that there was a by-pass for the air. During auscultation the patient was shaken by the interne but no tinkling sound could be elicited, but on questioning the patient he informed me that the bell had no clapper.

*Operation.* Jan. 4, 1917. Under chloroform drop anesthesia a Kahler bronchoscope was passed and a thimble-like body with its wide end uppermost was located firmly impacted in the right bronchus. A small hole, apparently 1-16 inch in diameter, through which the air bubbled, could be seen in the

distal end of the foreign body. Forceps of various kinds were repeatedly applied to the free edge of the foreign body, but they all slipped off since traction seemed to tilt the foreign body and force its sharp edge into the opposite bronchial wall. The end of a hooked probe was introduced through the hole in the "bell," and at one time I thought the foreign body had dropped into the pharynx as the bronchoscope was being withdrawn. To determine the exact position of the foreign body the patient was then taken to the X-ray room, but the fluoroscope showed the foreign body in its original position. Upon returning to the operating room a Jackson tube was passed without further anesthesia, but the forceps slipped as before.

At one time the child ceased breathing and a tracheotomy was begun, but the child reacted as the skin was cut. The operation was discontinued at this time, pending the manufacture of a special extractor. This was made on the following day, according to my design, and consisted of a long brass rod with a tapering screw thread at one end which was intended to be screwed into the hole in the end of the foreign body, on the principle of a bolt and nut. (Fig. IV.)

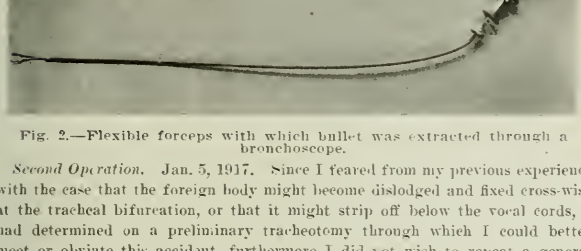


Fig. 2.—Flexible forceps with which bullet was extracted through a bronchoscope.

*Second Operation.* Jan. 5, 1917. Since I feared from my previous experience with the case that the foreign body might become dislodged and fixed cross-wise at the tracheal bifurcation, or that it might strip off below the vocal cords, I had determined on a preliminary tracheotomy through which I could better meet or obviate this accident, furthermore I did not wish to repeat a general anesthetic.

Under local anesthesia a low tracheotomy was made through which a short (9 mm.) Jackson bronchoscope was introduced and the foreign body located. Dr. Merrick McCarthy, who was assisting me, then screwed the special extractor into the hole in the foreign body, which was easily withdrawn with the bronchoscope until the tracheal opening was reached, when it was stripped off. I immediately seized it with a forceps and extracted it. It proved to be a brass umbrella ferule 15 mm. long and 10 mm. in its widest diameter. (Fig. V, B.)

*Comment.* These cases illustrate certain features of foreign bodies in the bronchi. In the first place physicians and laymen realize that foreign bodies can readily be removed and patients are no longer neglected, but are more frequently sent to clinics and hospitals.

The physical findings and roentgenograms were characterized in the first case for complete obstruction of a bronchus, while in the second, although the

foreign body was a large one, a by-pass for air could be diagnosticated. The necessity for improvising instruments to meet unusual conditions is especially well demonstrated in Case II. I find that Richardson, (1) devised an instrument resembling a corkscrew to remove a rubber eraser which plugged a bronchus, while the instrument which I employed may be likened to a bolt and nut. The use of a flexible forceps has some advantages when the tactile sense is employed in extraction, since there is but slight danger of traumatizing the tissues with such an instrument. Although Jackson's (2) contention is correct, that foreign bodies should be seized under ocular guidance, still in exceptional cases with

Fig. 3.—Roentgenogram showing umbrella ferrule in right bronchus.

proximally lighted tubes we must also depend to a considerable degree upon touch as well.

## II. SPONTANEOUS EXPULSION OF AN UPHOLSTERY TACK FROM THE RIGHT BRONCHUS.

On the afternoon of September 6th, 1914, while playing with an upholstery tack, a little girl, V. R., aged six years, lost the tack in a piece of cloth, and in shaking out the cloth the tack dropped into her mouth. She was immediately seized with violent coughing fits. Those about her inverted her and attempted to dislodge the foreign body.

Dr. Charles Heisel was called a short time thereafter, and he also attempted inversion, but found that this increased the coughing. At my suggestion (telephone) the patient was taken at once to the Jewish Hospital, and Dr. S. Lange made a roentgenogram, which showed the tack lodged head downward in the right bronchus, opposite the fourth rib posteriorly.

On examination there was slight coughing but no dyspnea. Impaired resonance and feeble breath sounds were found over the base of the right lung with squeaking rales chiefly on the right side. The child was given 1-200 grain of atropine and sent to the operating room. Before the anesthetic was administered I found that the most suitable forceps for the case was out of order. I therefore decided to postpone the bronchoscopy until 8 o'clock the same evening.

A short time after I had left the hospital, the interne, Dr. F. Goldenberg, telephoned me that the child had coughed up the tack. It measured 18 mm. in length, and the head was 7 mm. in diameter. (Fig. V, C.)

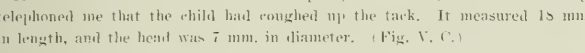


Fig. 4.—Illustrating bolt and nut principle of extraction of the ferrule.

A. B. C.

Fig. 5.—A, bullet, actual size, removed from bronchus (Fig. 1). B, umbrella ferrule, actual size, removed from bronchus (Fig. 3). C, upholstery tack, actual size, spontaneously expelled from bronchus.

*Comment.* Unfortunately, I have been unable to find the X-ray plate of this case, which proves to be a rather important one since Jackson (2) in his recent book states that a foreign body of this kind has never been expelled spontaneously.

From the increased coughing on inversion we may assume that the tack was at first free in the air passages. Additional factors favoring spontaneous expulsion were the short sojourn, (three to four hours) of the foreign body, the rest in bed; the administration of atropine which possibly relieved the spasm of the bronchus, which had been manifested by the squeaking rales and finally the tack proved to have but a moderately sharp point.

The manner of entrance as well as exit of this foreign body was certainly out of the ordinary.

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## DISCUSSION.

**Dr. J. W. Murphy,** Cincinnati, Ohio: The cases reported by Dr. Iglauer are very interesting. The point that he brought out as to the difficulty of removing a foreign body of this character after it has been in the lungs, was well emphasized in a case which I had last summer. The longer these bodies remain impacted the more swollen the tissues become, and the greater difficulty there is in extracting them. Last summer a patient of Dr. Bledsoe, who had been wearing a tracheotomy tube for a number of years, while walking along the street lost the stem of the tracheotomy tube. He went to the doctor's office, who at once sent him over to me. To my surprise, the patient was having no discomfort. There were no local symptoms, and physical examination was negative, so much so that I was doubtful whether the foreign body had been inspired, thought probably it might have dropped in the clothing some place. The stem was three inches long. There was not much difficulty in this case in making an examination, as we already had the tracheotomy opening, so a lower bronchoscopic examination was made, but I was not able to locate the foreign body. Thought it might probably be in the trachea. I then decided to see if the X-ray would give any information, but not being able to find any of the men in at that time, I decided to make another attempt, going below the bifurcation. I first explored the right bronchus, thinking probably it had gone in that direction, but with negative results. I then explored the left bronchus and found the foreign body firmly imbedded in it, so much so that I was not able to see it until the Jackson tube came down upon it. It had become encysted and so impacted in the lung as to become practically a part of the lung. There was no difficulty in breathing; no pain. I attempted to grasp the end of the tube. The mucous membrane was much swollen and overhanging the end of the tube. Also the flange end was up, and that made it much more difficult. It was only after considerable manipulation that I was able to grasp the tube and bring it up to the opening, when a new difficulty presented. The flange end was larger than the tracheotomy opening, and while attempting to grasp it with the hemostat it was loosened and fell back into the trachea. In a short time I succeeded in getting it again, and brought it to the opening, when it was grasped with a hemostat and held until the tracheotomy opening could be enlarged, when it was withdrawn. The interesting feature was that this large tube, almost three inches in length, and so near the size of the bronchial tube, should have formed really a firm part of the tube and cause no discomfort whatever. So much so that I was very much in doubt whether we had a foreign body until we were able to grasp it.

**Dr. G. W. Boot,** Chicago, Ill.: In this connection I would like to speak of a case which I reported some four years ago. Five years ago a boy eight years of age was given a toy bow and arrow for Christmas. He was holding the end of the arrow in his mouth when the head, a thimble-shaped object, was inhaled. Following this the boy had two or three attacks of pneumonia from which he recovered. He then was considered by his attending physicians as having developed t. b. His parents, who were Germans, took him to Germany. There he was examined by a couple of physicians who failed to find the cause of the trouble. He was brought back to America after the money was all gone, and taken to the Children's Memorial Hospital for an X-ray picture. The plate was developed and set up in the examining box. The boy and the physician, who happened to be in the hospital at the time, and the mother, went in to see the picture. Before the physician could say anything the boy said, "There it is mother," pointing to the foreign body in the picture. I was called then to see the case. I did a tracheotomy and searched for the foreign body, which was in the

left lung, but failed to find it. Passed the bronchoscope tube down the left bronchus, and examined all the branches but could find no foreign body. I waited a week, and then made another examination under local anesthesia through the tracheotomy wound, but again found nothing. After another week the examination was repeated under local anesthesia, but again with negative results. I noticed, however, that when I passed one point in the left bronchus the boy would invariably cough. There was a small patch of granulation at this point. I passed the probe into the left bronchus, into all the divisions I could find, without touching anything. I then probed where this patch of granulation was and felt a click, and the boy insisted that he heard it; so without seeing the foreign body I passed a pair of dilating forceps into the opening and the thimble-shaped object was caught in the branches of the forceps and withdrawn.

In connection with Dr. Iglauer's third case, I would like to mention the case of a young man who came to the Cook County Hospital two years ago, with a history of having eaten ice cream on January 1st, and swallowed a pin. This pin had been somewhere in his anatomy for about two months, when he came to the hospital. I ordered an X-ray picture so as to locate the foreign body, but before it could be taken the boy had a coughing spell and expelled the pin. In that case I think the pin had penetrated from the esophagus into the trachea. The boy had been coughing up blood before he came to the hospital. As soon as the pin had been expelled the patient thought his troubles were over and left by the back door, without telling us anything about it, and we saw nothing further of him.

In speaking of this case to one of my patients, she said that when a young woman she had inhaled a pin which had given no particular trouble, and several months afterwards she coughed it up.

**Dr. H. W. Loeb,** St. Louis, Mo.: The last case of Dr. Iglauer's reminds me of a case I had, with a somewhat different result. I assume in instances of this kind, the broad part being made of light metal is carried in first, and therefore expelled. The case that I had was that of a doctor who had a fat, short neck. Some of you who have had similar experiences can imagine what sort of a case it was. He came from the country. He had been having his teeth treated, and the dentist had lost one of his instruments. He had not had any symptoms, but he wanted a picture taken. The picture showed the dental instrument with the sharp end (it was not a broach) sticking in the trachea just above the bifurcation on the right side. I attempted to remove it under a general anesthetic, but failed because I was not expert. Had a good deal of assistance, but no one succeeded in getting it. He was having no symptoms, no cough, and I would not have known it was there except that the instrument had disappeared. I undertook to remove it the following day, but in the meantime another picture was taken, and this showed the instrument sticking in the right vocal cord. Before we undertook a second operation we had a fluoroscopic picture, and then found the instrument in the secondary bronchus, sticking up. I was not very anxious, nor was any one else very anxious to go down after that particular instrument, the feeling being that removal would rip open the bronchus. I told the Doctor that if it was my bronchus I would take it to Jackson. By the time he reached Jackson it had reached the tertiary bronchus. It took Jackson eleven seconds to get it out, without an anesthetic.

**Dr. Thomas Hubbard,** Toledo, Ohio: I think this question of anesthesia and tracheotomy is a good subject for open discussion. It should be decided on general surgical principles, and that a man should not feel any hesitancy, or feel that he is doing wrong if he does a tracheotomy and the same applies to the anesthetic. Tracheotomy helps to

straighten the bronchial angle and allows the operator to work through a charter tube.

Dr. Chamberlin brought up the question of anesthesia. I think this question is worthy of discussion, and in the end general surgical principles will prevail. I think that I do better work and more careful work under general anesthesia. And I would further in reference to anesthesia—Dr. Iglauer's case brings this matter up—avoid combined narcosis. The anesthetist works better with one anesthesia. While in an adult it is allowable to give small doses of morphine occasionally, even that should be done with great caution. In general avoid confusing the anesthetist by the administration of two or more narcotics. You may get the accumulative effects from them when you least expect them.

**Dr. Pierce:** You mean in case of a foreign body?

**Dr. Hubbard:** This applies particularly to any operation in the lower respiratory tract.

**Dr. D. W. Layman,** Indianapolis, Ind.: Since Dr. Loeb reported his case, I would like to speak of a similar case which I had in Indianapolis —a case of a dental reamer, accidentally dropped into the patient's mouth and inspired by the patient. I took the case over to Dr. Jackson, of Pittsburgh, and he very successfully removed the instrument, which was found in the secondary bronchi, well down. Dr. Jackson told me at the time that he was going to make a special report of these dental foreign body cases, namely dental reamers and broaches. It seems to me that a report of such cases would be of great value, especially in warning the dentists. I think there are quite a few of these cases that have not been reported, and if reported the dentists might be led to find some mechanical way of preventing such accidents.

**Dr. J. C. Beck,** Chicago, Ill.: Dr. Iglauer's paper brought out a point —that of transferring the patient from the operating room to the fluoroscopic room, then back again. It has been our custom to operate these cases in the fluoroscopic room. Of course the room must be large enough so you can do this work. Not only that, but the preliminary picture, which in children is necessary, or the fluroscopic examination for diagnosis is made under an anesthetic, and therefore the fluoroscopic room should be so constructed as to enable one to operate in this room. Make your fluroscopic examination and operate at the same time, thus avoiding the carrying the patient back and forth, which is certainly a taxation, a very great inconvenience, and a time-taking proposition.

The second point is—the brass tack and its spontaneous expulsion. Contrary to that, I had a case of a similar nature, in which we made two attempts and failed. The patient went up to Rochester. There they failed in the first attempt, but in the second attempt succeeded in removing the brass tack.

I was called one time to Wisconsin to see a case of foreign body in the bronchus. A boy, seven years old, was seized with a pain in the chest, and on inquiry of the boy what the trouble was, he pointed to the light of the gas fixture. They discovered that the gas tip was missing. The fear was that he inhaled it. The family doctor was called, he having at this time three cases of bronchitis and pneumonia in this family under treatment. I did not reach there until early next morning. Then the proposition was that a picture would have to be taken, but this became very inconvenient. I told the doctor we would wait until the next morning. The next morning we had a frank bilateral pneumonia. Fortunately it was inconvenient to get the X-ray apparatus, as the wait saved this child from manipulation and possibly death.

One of the first cases, and about the only case I ever had in which I removed a foreign body successfully from the right bronchus, was a case in which a miniature lamp had been inhaled. This case was operated upon early in the days of bronchoscopy. I made a tracheotomy and saw the foreign body. The patient was allowed to come out of the anesthetic somewhat, I tickled his throat with a probe, and he coughed the foreign body out.

A most interesting case of spontaneous expulsion of a foreign body was Ballenger's celebrated case, in which the foreign body was deep down in the larynx. The child was brought from Mexico in a special car. The doctor cocainized very thoroughly and he and I could see the foreign body, through the swollen mucous membrane of the larynx. The patient was ordered transferred to the hospital for the purpose of removal of the foreign body. On the way to the hospital, over the jarring pavements of Chicago streets, the child coughed and expelled the foreign body, which was a piece of a palm leaf fan.

**Dr. Iglauer** (closing the discussion): It is growing late and I shall not talk very long.

Dr. Murphy brought up the question of swelling around the foreign body. I think that can be somewhat relieved by applying adrenalin to the mucosa, possibly adding a little cocain. He also brought out the fact that it was very hard to see a hollow foreign body through the bronchoscope. We had this same difficulty with this ferrule, which fitted so tight that the most prominent thing to my eye was the hole in the end of the ferrule. One is apt to look through hollow foreign bodies.

The question of tracheotomy has been touched upon by several speakers. I think we all hesitate to do a tracheotomy because Jackson gets along without one; but the average man of more limited experience, if he feels that conditions are such that he can succeed better with a tracheotomy he had perhaps best not postpone it too long. I do not believe that tracheotomy in itself is a very dangerous operation. Most tracheotomy subjects recover unless they have diphtheria, or something of that kind as a complication. Jackson of course contends that you do not get a better angle by doing a tracheotomy; that by moving the patient's head to the left or right one can get in the opposite bronchus as easily as one can through a tracheal incision. There is one advantage: one can introduce a larger tube through the trachael incision. In the boy, 12 years of age, we were able to get down a No. 9 Jackson tube. We could not have introduced it through the mouth. On the other hand, Jackson points out that the small tube has certain advantages, as it permits of more liberal movement, the small bronchoscope can be moved from side to side, but the large one cannot. Possibly in the case of the ferrule, had I had the special extractor I might have pulled the foreign body out through the mouth as easily perhaps as it came through the trachea, but there was certainly great danger of its slipping off and falling back. I think in this case we were justified in using the tracheal incision.

Dr. Hubbard brought up the question of anesthesia after administering narcotics and other drugs. I do not believe the doctor would object to the administration of atropin, especially as he comes from that part of the state where it is lauded as the great preventer of shock, as worked out by Crile.

**Dr. Hubbard:** With atropin you lose the pupil reflexes as a guide in the work.

**Dr. Iglauer:** That may be, but it has the additional advantage of drying up the secretions, which is a great aid. Now it is questionable whether morphin or codein should be given. Jackson objects because it does away with the cough reflex. Killian advises codein before doing suspension laryngoscopy in children. According to Hewitt the

ether chloroform sequence is safer than chloroform from the start the chief danger of chloroform being during the first stages of anesthesia.

Dr. Beck spoke of the advantage of having the fluoroscope in the same room in which you do your operating. The Doctor has been in the Cincinnati Hospital and will recall that the X-ray room is in the basement. We have no trouble in taking the patient down on the elevator and bringing him back. It only takes a few minutes. He thought I was compelled to desist because I took the patient to the X-ray room. That was not the case. It gave me a much needed rest, and also saved time in searching for the foreign body which possibly had slipped down the esophagus. The patient was brought back to the operating room, but given no more anesthetic and the operation continued until I decided that he had had enough manipulation for that day.

In this particular case in which I did a tracheotomy, we were bothered with a peculiar form of bleeding. The child would bleed only on expiration or on coughing. We could not see where the blood came from. It seemed to come from under the muscle. We finally went ahead and ignored the hemorrhage. The hemorrhage ceased when the bronchoscope was put in, but after the foreign body was out he began to bleed again. We enlarged the incision to one side, and found a vein which had been bleeding, and had given us this intermittent hemorrhage.

# REPORT OF SIX CASES OF DYSPHAGIA AND DYSPHONIA OF DENTAL ORIGIN.

By THOMAS J. HARRIS, M. D., New York, N. Y.

Among the many recent advances in medical science none is of more importance than that which has been so forcibly brought to the attention of the profession in the last few years by the work of Rosenow and Billings in showing the important role that focal infection plays in the production of systemic disease.

We as laryngologists have had this impressed upon us in the many discussions which have taken place with the tonsil as a battle-ground, until today there is little dissent from the opinion that the diseased tonsil is, if nothing more, a decided menace to good health. It is important, however, to constantly bear in mind that the tonsil is only one possible cause of infection and that in determining the sources of systemic disease every possible centre must be considered. The danger of falling into error by overlooking this fact has been shown by our fellow-member, Dr. Charles W. Richardson, who, in a paper a short time ago, reported a number of cases where the tonsils had been removed for the relief of systemic disease and later the cause of the trouble was found to reside elsewhere, as well as a second group where the patients were sent to him with the request that the tonsils be removed for the relief of certain trouble and where he was able to demonstrate the existence of a focus of infection elsewhere.

Of equal importance as a cause of infection with the tonsils is disease of the teeth. In spite of the admirable work of Hartzell and others there is still a surprising ignorance of the subject on the part of physicians and especially of dentists. Great credit is due to the educational campaign which the worthy secretary of this Society has been carrying on to enlighten both physicians and dentists of the deadly peril that lies in the apical tooth abscess. In a dense sea of ignorance his has been almost the only voice raised to urge that due weight should be paid to this important source of infection.

It is truly amazing in spite of all that has been written and said how densely ignorant, or what is even more reprehensible, how indifferent the majority of dentists are to the subject. The

attitude of one prominent dentist in New York, honest in every respect and doing according to the accepted dental standard the highest grade of work, is characteristic of the great mass of the profession. In a recent conversation with him he did not hesitate to declare that the whole subject was purely a fad on the part of dishonest or visionary dentists and that all the present craze about pulling teeth was nothing less than criminal.

We do not desire to enter at further length upon this subject, but our own strong feelings over it lead us to say what we have prefatory to the report of a brief series of cases illustrating what to us is an altogether new manifestation of focal infection proceeding from the teeth.

*Case 1.* S. G., age 40, was first seen June 21st of last year complaining that for five days he had had difficulty in deglutition. There was no history of a foreign body, no pain in the throat. Fluids went down easily but it was difficult or impossible to swallow solid food. Examination of the throat was negative. There was no inflammation of the pharynx, larynx or mouth of the œsophagus. No direct examination was made. Patient was a schoolmaster approaching the end of the school year and naturally more or less tired. Strychnine was prescribed and the patient was instructed to report in two days. When next seen the difficulty in swallowing had materially increased so that even fluids were gotten down with some difficulty. There was also great fatigue in talking so that the patient found it trying to speak above a whisper. A second examination of the throat revealed no inflammatory condition. At this time attention was particularly directed to the teeth which were found to be extensively gold-capped. Based upon our previous experience with the teeth this was regarded with suspicion, and an X-ray picture was ordered to be taken at once. This revealed abscesses at the apices of numerous of the gold-capped teeth. Immediate extraction of the abscessed teeth was advised. This was performed the eighth day of the disease. A culture made from the pus of the teeth showed streptococcus viridans. Rapid improvement in swallowing took place and in two weeks time the difficulty in swallowing and speech had entirely disappeared. The patient during the brief interval between the onset of the attack and the surgery of the mouth had lost a considerable amount of weight which required a number of weeks to be restored.

*Case 2.* A. D., age 33, was first seen July 21st with the complaint that for one year he had had difficulty in speaking and in swallowing. Patient was a man of normal size, gave no history of previous illness and there was no suspicion of any central lesion. The condition of the voice attracted attention at once. There seemed to be little power over the laryngeal and pharyngeal muscles. It suggested decidedly the voice of a child suffering from an attack of post-diphtheritic paralysis of the soft palate. Examination of throat and larynx was entirely negative. The patient's occupation was not one that demanded much voice use. Formerly he had been a choir singer but for the last year had to give it up. Pyorrhea alveolaris was discovered. An X-ray of the teeth showed no apical abscess. Careful treatment of the pyorrhea was begun. Immediate improvement in the symptoms followed. The pharynx, voice and

swallowing steadily improved and at end of the two months he was virtually well.

*Case 3.* Mrs. J. C., age 32, was first seen August 21st, complaining of an obstruction in the throat. She was exceedingly nervous and could not sleep; examination of throat was negative. Diagnosis of hysteria was tentatively made and internal medication ordered. Some improvement followed, but not pronounced. At second visit attention directed to teeth; an X-ray of teeth was ordered. Abcesses at the apices of four teeth were found and the teeth extracted. Streptococcus viridans was discovered in pus. An autogenous vaccine was administered. Great improvement took place. Patient at the end of three months discharged well.

*Case 4.* Hospital case, young man, age 20, presented himself complaining of inability to swallow. Examination of throat negative. This case was examined with the esophagoscope with negative findings. Patient showed evidences of dental abcess. An X-ray was ordered and the diagnosis confirmed. Extraction of abscessed teeth was performed with prompt and satisfactory relief of symptoms.

To these we are able to add by the courtesy of Dr. Haskin the reports of two other cases of a similar nature.

*Case 1.* F. C., age 15, was referred to Dr. Haskin for loss of voice which had extended over ten days. A diagnosis was made of hysterical laryngitis. Patient was pale and seemed mentally depressed; poor appetite. Adenoids and tonsils had been removed. Examination of teeth showed the upper six year molars to be in bad condition and painful upon pressure over the roots. Radiograph proved them to be badly filled and with infection at the apices. The fillings were extracted with the escape of offensive gas and secretion. Prompt recovery of the voice took place.

*Case 2.* Mrs. M. F., age 62, presented herself on account of great difficulty and pain in swallowing. There was a history of rheumatism and bad heart. Examination showed a small pocket of pus in the left tonsil, seven badly diseased roots with marked pyorrhea and much pus; blood pressure, 230. The pus pocket in the tonsil was opened and drained without relief to the symptoms. The entire seven teeth were then extracted. Immediate improvement followed with little pain for over a week. With the exception of a single application of nitrate of silver to the lingual tonsil no further treatment was given. Two weeks after the extraction of the teeth patient reported being entirely free from all difficulty or pain in swallowing and feeling better than in years. Blood pressure continued to drop until two months after the operation it had fallen to 185.

The clinical history and outcome in these six cases have been of more than ordinary interest to us.

The condition of the first case, S. G., when first seen was such as to give us decided concern. He was an old patient who had not been ill for years and not given to complaint. His inability to swallow as well as talk when first seen had already begun to affect his general health and the pain and apparent paralysis of the muscle of swallowing and speaking rapidly increased in the short time elapsing before the true cause of the symptom com-

plained of was realized and the proper treatment instituted. He was totally unaware of having any trouble with his teeth. Examination of the mouth showed extensive bridge work in both the upper and lower jaw. The X-ray picture revealed large abscesses at the apices of more than eight teeth. Equally rapid with the onset of the symptoms was the relief secured by the removal of the diseased teeth.

In case two the symptoms were not so pronounced but had extended over a much longer period of time. As stated in the report of the case, the character of the voice was peculiar and attracted immediate attention upon his entering the consultation room. It was first strong but almost immediately weakened and suggested a partial or complete loss of function of the pharyngeal muscles. Here the trouble was not one of apical abscess but of extensive pyorrhea. We employed here, after the proper treatment had been given to the teeth, an autogenous vaccine over a considerable period of time. The organism was the same as is wont to be found in most of these cases, streptococcus viridans.

Case three was also a chronic case with many of the symptoms of what we are apt to pronounce those of hysteria. Diagnosis of diseased tonsils had been made and an operation recommended although there was almost no tonsil present. Here also the use of an autogenous vaccine in connection with dental and other treatment effected satisfactory result.

Case four was more acute in its onset. No history could be elicited of foreign body nor the swallowing of an irritating fluid. His complaint in regard to swallowing was so pronounced however that we felt warranted in making an endoscopic examination which, of course, revealed nothing. Serious trouble was found in this patient's mouth and proper treatment gave prompt relief to the dysphagia. Just how the infection worked in this case it is not possible to positively state. It would seem most probably there must have existed, as in the better known conditions affecting the heart or other organs of the body, an actual toxemia of the muscles concerned. While this form of toxemia is entirely new to us there is little question that it is not a rare condition and will explain many cases that have been puzzling us and which we have been regarding as pure neurosis. In this connection allow me to speak earnestly of the importance of not depending upon a mere examination of the teeth by any dentist, however competent he may be, but that every case should have complete and satisfactory

X-ray films made. These, moreover, should be interpreted by one who is accustomed to do so and not left to someone entirely ignorant on the subject.

We as specialists cannot too soon get away from the practice of depending on as final the opinion of the family dentist that there is nothing the matter with the patient's teeth. However good a dentist may be no examination by him can take the place of a good X-ray picture and where such a picture reveals the presence of an abscess we are yet to be convinced that any dentist can drain the abscess satisfactorily. Certainly any such attempt should be followed up by another roentgenogram.

## DISCUSSION.

**Dr. J. M. Ingersoll**, Cleveland, Ohio: The paper brings before me a subject which we are all thinking about more or less, and emphasizes very markedly, how careful we should be in making a diagnosis. The opinion of a dentist in regard to the teeth is necessary but should not be accepted as final until a Roentgenogram of the teeth has been made.

One of the cases cited in which there was absolutely no indication, so far as the patient was concerned, of any trouble in the teeth, also brings out a point more or less frequently seen, namely, that teeth which may seem to be above suspicion, particularly in obscure cases, should be carefully considered and roentgenograms made of them. We know that the teeth are frequently found to be at fault in obscure cases.

We should be on the lookout for these symptoms in obscure cases, and the variety of symptoms which may come from trouble around the roots of teeth should be constantly in our mind.

Tonsils are frequently the cause of trouble, and yet we sometimes do a tonsillectomy after excluding other causes of trouble, and are disappointed because we do not get results. I question whether we are always as careful as we should be in excluding other causes. On the other hand, we all occasionally see tonsils which show comparatively little evidence of much infection in them, even after a careful examination, but when we section them, after removal, we find pockets of pus deep down in the tonsillar tissue and thus know that we were justified in removing them.

**Dr. J. A. Thompson**, Cincinnati, Ohio: Dr. Harris' paper recalled a case which showed that symptoms in the throat may not alone be due to bad teeth, but that sometimes our apparent bad results in other regions may be due to unsuspected teeth. I operated upon a patient a year ago for the relief of ethmoid headache. She was six feet tall and weighed 180 pounds. She was the picture apparently of perfect health. She had been treated in Philadelphia, New York, London, Paris, Berlin and Vienna, for chronic headaches, but without relief. She was referred to me by another patient whose headaches I had cured. I drained the anterior ethmoid cells, and got a perfect result. Six months later she came back complaining that the symptoms had returned. She had each afternoon a slight rise in temperature with headache and pain in the frontal regions. The operation had been a rank failure according to her. Examination of the nose showed it to be in as perfect condition as I ever saw a nose after operation. I began to investigate elsewhere for the source of toxemia. The first molar in the right side of the upper jaw excited suspicion, and I asked for an examination by a dentist. The dentist reported that the tooth was in perfect condition.

I objected to the dentist's opinion because of the color of the tooth and asked for an X-ray. There was an abscess at the apex of one root. Extraction of the tooth cured the headache, and I am again in the good graces of the patient because the headache is gone, and my operation a success.

**Dr. J. C. Beck**, Chicago, Ill.: As Dr. Harris read his case reports I classed them—Case 1, Hy., Case 2, Hy., Case 3, Hy., and so on. I do not mean to imply that these cases were hysterical, and the removal of the teeth cured the hysteria, and yet I begin to wonder whether it is not so. What is hysteria? It is a psychosis—a weakened condition of the muscles. Is it not possible that this toxemia which comes from the teeth may cause this weakness, resulting in what we call hysteria? We get this toxemia from blind abscesses. It is a fact that the dentists have been very conservative about saving bad teeth. One tooth is worth a good deal, and they would not consent to attack these apical infections. They would have nothing to do with roentgenograms. I have had dentists say to my patients—"What you need is a good nose doctor. You don't need a dentist. Your teeth are all right." Yet, there were blind abscesses causing trouble. Now the pendulum has swung the other way. Almost everybody's trouble is brought back to the teeth, and bridges are pulled down, and teeth are drawn, or extracted, or whatever you wish to call it, for the relief of so many conditions that are not cured. Now it seems to me that the last word our President mentioned should not be taken too seriously—that these abscesses cannot be drained. It has been an old practice, and is still practiced by many dentists, to resect the roots of these apices, and drain the infected area. When we see a little dark area in the bone, it is a sign of infection and the tooth should come out. If it is found in the roentgenogram that the filling has not gone clear up, the tooth is condemned and should be drawn. Yet it is perhaps serving a very good purpose, may be even holding a bridge, but there is that little area and the tooth is condemned. As a matter of fact this area is very easily explored, it is being done, and the cavity allowed to heal by granulation. If the symptoms disappear that is what you are after, and you have accomplished your purpose. I think as much as they were formerly opposed to the removal of teeth, the dentists are now overly anxious to remove teeth, because the general men have pointed out to them the dangers. Dr. Gilmore has stated that more people in Chicago die from imperfectly filled teeth than from practically any other condition. He is broad in his statement, but he says the amount of disease due to imperfectly filled teeth, and infected apical areas is enormous. I have now something like forty cases of this type in which the root has been resected, or in which the dentist has simply removed the bone above the tooth and obliterated it, using a bone wax plug after cleaning the area out—the wax that is used in bone surgery. It stays in until it is extruded, and healthy bone fills in the cavity. It is a question whether this should not be tried first before advising the removal of teeth, and then not getting the result.

**Dr. Harris** (closing the discussion): Mr. Chairman, I will only say a word in reply to what Dr. Beck has said. He has opened up a very broad, and to me a very interesting subject. There is no great advance, wherever we may look for it, where there is not danger of the pendulum swinging too far in one direction or another. I freely admit that so far as these focal infections are concerned there is this danger in regard to either the tonsils or the teeth. I feel, however, that where one dentist may be accused of doing this, there are fifty who are totally indifferent on the other side. There is considerable in what Dr. Beck has said, and I agree with him, certainly in theory, as to the first thing which we must establish beyond peradventure, if pos-

sible, and that is, that the systemic trouble is proceeding from the teeth.

I wish, however, I could follow Dr. Beck in his enthusiasm as to the ability of not the average dentist, but the great majority, to do successfully the work he speaks of. I have in mind the extraction of the tooth, versus the removal of the filling, then treating through the root canal the apex of the tooth, the obliterating of the diseased area; that is what in my mind is almost impossible of successful accomplishment, when you consider how absolutely difficult it is to get a sterile wound in that root canal: how almost impossible for a dentist to succeed in introducing a perfectly sterile filling. Then after continuing the work, extending many times over a period of weeks, and the patient has suffered a great deal of pain in an effort to save the tooth, it is frequently found that the tooth has to be extracted. I do not believe we should be reckless in advising the extraction of teeth. I speak of it feelingly as I do, because I do not view my own case with optimism. I did not know that I had any trouble whatever with my teeth, and yet I was shown to my own satisfaction that I have at least four apical abscesses. I have no systematic trouble whatever. If I felt encouraged that dentists could work successfully along the lines indicated by Dr. Beck, I would advise patients accordingly, but I feel where one man might be able to do this work, the great majority of dentists to whom cases are referred would find it impossible, or if they attempted it would make a rank failure.

# FURTHER REPORT ON THE TREATMENT OF MALIGNANT DISEASE OF THE LARYNX.

1. Percy coagulation method.
2. Diathermia coagulation method.
3. Deep X-ray penetration.
4. Massive radium application.
5. Laryngectomy.

By JOSEPH C. BECK, M. D., Chicago, Ill.

In making this further report on the cancer question as it refers to our Specialty and the larynx in particular, I wish to call your attention very briefly to my last report read before this Section last year in St. Louis, and published in the Laryngoscope 1916. In that report I stated that we had not yet any specific treatment or cure for this terrible affliction, that the surgical treatment when done thoroughly and immediately after an early diagnosis gave the only chance. I also described other than surgical methods of treatment, such as diathermia, deep penetration Roentgen ray, and particularly the Percy coagulation method of removing the neoplasm. In the summary of cases treated by these methods I stated that the Roentgen rays aided by the action of the diathermic current caused marked changes and often disappearance of the growth only to return and terminate fatally. That with radium in the dosage of ten mlgrs. pure, which I had at my disposal, no appreciable influence on the growth could be seen, but that the Percy coagulation method promised to do more than the X-rays or radium, as I then employed them.

Since the above I have continued to employ these various methods and have already made a brief report on the Percy coagulation treatment before the Chicago Medical Society this winter, which I will present again in this report, particularly in so far as the larynx is concerned.

I have also added in this report the use of the diathermic current, surgically, that is the mummification of the tissues, and the use of massive doses of radium. On these last two methods of treatment I wish to make only a preliminary report, because not enough time has elapsed to show their value as a permanent cure.

Finally I desire to report on laryngectomy, in one case in particular.

In the discussion of the cancer problems, particularly in the treatment, it has been the attitude of those taking part in the discussion to be very charitable, because all recognize the hopelessness thus far, in the advanced cases or deep-seated cancers. This attitude would be all right if it were not for one fact, and that is, that the essayist so often oversteps the boundary between enthusiasm and accuracy in making his report, thus promising too much. The result from this sort of a thing is an influx of material of cancer cases from physicians as well as the laity. The disappointment as a rule which follows to the doctor who sends the patient, as well as the family of the patient, and the loss of confidence in subsequent work along this line, is self evident by the lukewarm action when another report along the line of cancer is made by someone else.

The writer is cognizant of this fact from a recent experience not along the line of treatment, but in the study of the etiology of cancer. To further illustrate my point of the apparent indifference that the medical profession in general has in the cancer problem, because of the exaggerated statements made by most of the workers, I will very briefly narrate this experience. A laboratory student of the cancer question was given a fluid to examine obtained from the interior of an abdominal cancer. The smear showed a torulla. Culture made from this fluid and subsequently examined showed a rich brick red colored growth which was a pure culture of these same torullae or yeast cells. A quantity of these yeast cells were injected into mice and as a result there developed tumors which when examined by microscope showed all the evidences of epithelial cancer and invariably resulted in the rapid death of the animal. From these tumors in the animals the same growth of torulla were again obtained and when these were reinoculated into mice produced the same growth, namely, an epithelial mass. A great deal more detail work was done by this laboratory worker as well as by Drs. Carl and Emil Beck, and myself who aided and cooperated with him from the surgical and clinical side, in order to assist in possibly adding even a mite to this hopeless condition of cancer.

He (the laboratory worker being Dr. M. Buchsbaum, Chicago,) made his report before the Chicago Pathological Society after nearly three years of labor, and his efforts were received with little favor or perhaps I might say with indifference. Desirous of having his

work published in recognized Journals devoting their pages to strictly scientific subjects, particularly in the cancer problem, he asked for that privilege of the Editors of said Journals. What was the result—after reviewing his work and reading his manuscript it was returned with an indefinite statement, saying that it was not a paper they could accept. Is that not an indifference and a sad commentary on our American Journals? An example of credulity is the following:

A surgeon reports before the Chicago Medical Society on the value of bacterial enzymes injections in cases of cancer of the abdominal cavity, with cures. He cites a case of retroperitoneal tumor supposed to be sarcoma of considerable size, that was treated with injections of these enzymes and cured. There was no microscopical examination made; the father of the patient being a prominent surgeon, happy in the results, discusses the case at this meeting and accepts the fact that because the tumor disappeared the injections cured the sarcoma. Is that not credulity in the extreme, and to think that there was no dissension from these extravagant claims in the discussion that followed. The same is true of reports by other methods of treatment of cancer as X-ray, radium, mesothorium, diathermia and all sorts of injections of sera, solution of chemicals and what not.

In compiling the reports of the many methods employed outside of surgery in cases of cancer for the past twenty years or since the advent of X-ray as a therapeutic measure, I found not a single well-founded and authentic report of a cure of a cancer by such methods, although many beneficial results and improvements were cited. My personal results from experimenting with autolytic solution injections as well as radium, deep X-ray and diathermia therapy in carcinoma, have been of no permanent value. Surgery, which up to date is the only means of combating the disease, is too often defeated of a cure by recurrence, and here too frequently too early reports of cures are misleading, that is to say, the cases are not followed up long enough. The recent organization of the Society for the Prevention and Cure of Cancer has a wonderful opportunity in regards to the many problems that come up in the subject of cancer, and the control of these reports is not the least important.

The reasons why surgery is so often defeated may be stated as follows:

(1) The patients present themselves too late for us to make an early diagnosis.

(2) Failure to make an early diagnosis.

(3) Timidity in operating extensively, especially in the removal of the tributary glands.

(4) Implantation carcinoma along the field of operation.

All four of these reasons are more easily overcome in cancer of the larynx, because the earliest development of a neoplasm is called to the attention of the patient as well as the doctor by (a) hoarseness in an older individual, particularly in men, that does not disappear with or without treatment within two or three weeks. This should be looked upon with suspicion and carefully watched and examined for cancer. Not that every hoarseness that lasts that long proves to be cancer, but it must be ruled out. (b) This hoarseness brings the patient early enough for an early diagnosis. (c) Cancer of the larynx is usually found in the anterior portion of the cords, and from this region the lymphatic distribution is very small and secondary glandular involvement is a long time in developing. As Crile has so aptly stated, "It's a cancer within its box," consequently the second and third reasons do not apply, namely, early made diagnosis and no very extensive surgery is necessary, that is, no dissection of tributary glands. (d) Of course the removal of the larynx, which is the only means known today of positively insuring against implantation recurrence, is an extensive and to the patients a most depressing operation, because they believe that they will not be able to speak except with a buccal sound. Laryngectomy is also associated with considerable risk to the patient's life from the complications as shock, pneumonia and mediastinitis.

In order to overcome some of these great objections to the complete removal of the larynx as well as the recurrence, namely implantation, I have developed the technic of laryngeal fissure and removal of the neoplasm by means of the Percy coagulation method.

1. *Percy Coagulation Method.* This method of treatment was first recommended by Percy for inoperable uterine cancers, but subsequently also for operable cancers of the uterus as well as other parts of the body. I have employed the method for cancer of the nose (carcinomas and sarcomas), carcinoma of the upper jaw, lower jaw, thyroid gland, external ear and larynx, in twenty-three cases, as follows:

| | |
|---|---|
| Sarcoma, intranasal | 1 |
| Carcinoma, extranasal | 1 |
| Carcinoma of the upper jaw | 7 |
| Carcinoma of the upper and lower jaw | 2 |
| Naso, oro and hypopharynx | 1 |
| Lips | 2 |
| Thyroid gland | 1 |
| External ear | 1 |
| Tongue | 3 |
| Larynx | 4 |

It is not my purpose to go into the philosophy of the Percy coagulation method, because the detailed description may be found in several of the articles written by Dr. Percy and published in the Journals of the A. M. A. in the past three years, nor do I wish to describe in detail all the cases treated by me. All I wish to say is, that it is a slow method of destroying the carcinoma or sarcoma in situ by a sort of cooking of the tissues and subsequent sloughing or discharge. The ultimate cicatricial formation and tenacity of the necrotic mass are the objectionable features that I have found. As to complications which have been reported, that is ulcerations of the deodeum, I have observed none. I have had one case of larynx carcinoma in which a large thyroid gland was present, which was in the way and had to be partially resected and many ligatures applied, in which a hemorrhage took place on the tenth day, most probably from a sloughing of part of the gland. This hemorrhage rapidly filled up the patient's lungs and choked her before help could be summoned. The reasons of substituting a laryngotomy and removal of growth by the Percy method for a laryngectomy are:

1. The patient will consent more readily to an operation when he may be promised that he may have a voice even though it will not be normal.

2. He may even hope to have a normal breathing tube, that is not have to wear a permanent tracheotomy tube.

3. The operation is not as dangerous as a laryngectomy.

The real value of this (Percy coagulation) method of treating cancer of the larynx will be further brought out in the case reports that follow:

11. *Diathermia Coagulation Method.* Nagelschmidt in his text book which appeared about four years ago has given the technic of the surgical dealing with malignant disease by the use of diathermia coagulation method of treatment.

The principles underlying this treatment are the passing of high frequency current from one pole to the other, between which there is a portion of the tissue to be penetrated by this current. Usually about one centimeter of tissue is treated at a time. The radiation passes from a pointed electrode to a flat one, thus causing a conical destruction.

It is necessary to anesthetize the patient either by general anesthesia or by the use of novocain infiltration, because the treatment is very painful. A quantity of electricity from 800 to 1800 miliamperes is passed through, when one observes a sizzling noise and a vapor escaping from the tissue thus treated. Sometimes when the height of the degree of charge has been reached, an arc forms about the electrodes which is evidenced by a sharp report spark.

I have measured the degree of heat of the tissue after the treatment and have found that it varies from 110 to 130 degrees immediately after the current is turned off. A number of points are thus attacked at a time and the interval between the treatments depends entirely upon the affect of the pain on the patient. We have usually applied this treatment every other day.

The resultant destruction can be seen in the Figures 1 to 7 of the case reported, during a period of six weeks.

At no time is there any evidence of an odor as from a slough in other types of necrosis.

Another important factor is the control of bleeding by this method. The strangest observation that I have made is, that in spite of the marked destruction and excessive heat produced, the internal carotid artery, jugular vein and the facial nerves which were in close proximity to the region thus treated have not suffered loss of their function.

III. *Deep X-ray Penetration.* As far as the deep penetration X-rays are concerned we have practically discontinued its use in carcinoma of the larynx, owing to the fact that the one case of carcinoma of the larynx which I was banking on as cured of its intrinsic growth by this deep X-ray treatment, after two years came to laryngectomy and determined the futility for us of this kind of treatment. Quite different is this type of X-ray treatment as far as sarcoma is concerned. Here the value of this treatment can scarcely be estimated. While I have only one case of sarcoma of the larynx to report in which deep penetrating X-ray has been employed with apparent success, I have several cases of maxillary, postnasal and pharyngeal sarcoma which responded beautifully. (Note concluding remarks in discussions.)

IV. *Massive Doses of Radium.* Recently we have made use of massive doses of radium in malignant disease of the upper respiratory tract and in one case of sarcoma of the larynx. The amount employed was 125 mlgrs. pure radium for a period of 36 hours, rented from the Physicians' Radium Association, Chicago. What the ultimate result will be in this case I am not in a position to say, because it is too recent since it has been employed, but thus far I have seen a marked reduction of the growth.

V. *Laryngectomy.* Before this section in Detroit, February, 1914, I made a report on this subject, having operated eleven cases at that time. Since then I have not done a laryngectomy, principally owing to the high mortality rate and loss of voice, until very recently, and to that case I have referred when speaking about the failure of deep penetrating X-rays. This case is described later in detail. I satisfied myself with less radical measures such as described above. In so far as the value of the less radical surgical measures, that is laryngotomy and excision, I still have two cases one four years and the other nine, since operation, that have not had any recurrence. That of course is a small percentage compared to the fatalities from such a procedure.

Laryngectomy so far as the patient is concerned is much dreaded owing to the loss of the voice, and if an artificial larynx or means could be established where a substitute for the normal voice even though it be imperfect, could be promised, this serious objection would be greatly overcome. To this end I have recently been working and believe that I have succeeded in modifying the usual technic of laryngectomy so as to enable me to place such an artificial means from trachea to mouth, which promises to give satisfaction.

This too I will describe in detail in connection with the case report.

## CASE REPORTS.

*Case 1.* Mr. R., 54 years old, married, three children, many years a waiter and in the saloon business. Has always been well except for hoarseness, until eight months before presenting himself, when he noted considerable more hoarseness from which he never recovered, in spite of treatment by specialists. He had received thorough antiluetic treatment on general principles and as a therapeutic test, without any results. This hoarseness soon developed into a marked type with some embarrassment in respiration. He had lost in weight, but had no pain upon swallowing.

*Examination.* Somewhat emaciated looking man, but bodily examination shows him to be fairly muscular and to have plenty of fat. The physical examination was negative. Blood examination showed a two plus Wasser-

mann, but the Abderhalden was negative as to carcinoma. Sputum negative as to tuberculosis.

*Nose.* Indicative of a sinus trouble or atrophic rhinitis.

*Larynx.* Considerable difficulty in inspiration, making quite a marked noise, and very hoarse voice. Inspection revealed a thickening of both cords, particularly the left. It appeared as though there might be considerable thickening subglottically. The diagnosis pointed to a luetic condition with a possibility of a rhinoscleroma. I determined to do a suspension laryngoscopy in order to examine below the cords and also take out a little bit of tissue for microscopical examination. While doing this suspension or following the action of the cocain locally, the patient became suddenly very short of breath and cyanotic, so that I was compelled to do a hurried tracheotomy. That was all the treatment for about two weeks. He had now recovered, in fact gained some in weight. It was now possible to make this suspension and remove the tissue. There was considerable thickening below the cords and the removal of a bit of this tissue showed it subsequently under the microscope to be a squamous celled carcinoma. One week later a laryngotomy was performed and the growth removed from within the larynx in the following manner:

Under ether anesthesia through the tracheal canula, an incision was made with the ordinary instrument down to the thryoid cartilage.

The parts were widely retracted. By means of the Percy cautery the thryoid cartilage was seared in the median line. An incision was made through the cartilage with Jackson's scissors. The thryoid cartilage was now split, and widely retracted. By means of Percy's small knife the entire intralaryngeal mass was cooked without any cooling externally, or within the esophagus. This was accomplished by periodically lying this heated point against the tissue and waiting until one heard a sort of a hissing sound, but never permitting the tissues to become charred or burned. This procedure required about thirty minutes. It was found that some of this thickened tissue appeared to go down into the cricoid region, so concluded to communicate the tracheal opening with the laryngotomy, also by the aid of the Percy knife. The interior of the cricoid and tracheal region was similarly treated by the Percy heated point as above. An up and down Jackson tube was now put in and the larynx dressed wide open to promote the throwing off of the slough. The patient had not much of a reaction, in less than two weeks was fairly clean. At the end of one month the tube was left out and the wound allowed to close.

For about three weeks the patient breathed well and had a very fair voice, then he began to show evidences of breathing with some difficulty, and at the end of two weeks he had to be reopened, which was very easy, and a tracheotomy tube inserted. It was decided to wait, to ascertain if there was going to be any recurrence before attempting further treatment, and to establish normal breathing through the larynx. After about six weeks' observation, and showing no evidence of recurrence, I decided to employ an intubation tube, which has given me excellent service in treatment of laryngeal strictures from other causes. A piece of tissue removed for microscopic examination proved again to be negative. This tube was allowed to remain for about two weeks, when he showed some evidences of pain in swallowing. On removing this tube I found that it had produced a decubital ulcer on the lower posterior wall of the trachea. The result from this intubation was very satisfactory but the laryn-

gotomy wound was kept open nevertheless by means of sounds in order to observe any recurrences.

Conclusions in this case, in October, 1916, were:

1. That he is possibly cured from carcinoma of the larynx.

2. That Percy treatment without cooling apparatus produces greater cicatrization than when it is employed, as will be shown in a subsequent case.

3. That there may be an element of lues in the case, that is to say both diseases may be present.

4. That tubes should not be left in situ without examining for possible decubitus ulcerations from pressure. The result from this mode of dilatation is, however, not promising in this case, because, after a few moments, the larynx opening closes down and the tracheotomy tube must be again inserted. It is my opinion that he will have to wear an up and down tube or an intubation tube for a much longer period and if after that it will not remain open, then he will have to wear a permanent tracheotomy tube. In that case he would be given one that he could close by the aid of the shuttle, operated from his pocket.

*Subsequent Course.* About one week after the above date (Nov. 16th) the patient showed evidences of great difficulty in swallowing and marked symptoms of septicemia. On exploration I found the posterior laryngotracheal surface in a state of coagulation necrosis. A day or two later the patient showed evidences of a fistula between the esophagus and the trachea, in the midst of this necrotic tissue, so that he was put on rectal feeding. It was soon found that he could not subsist long on this method of feeding, so I advised a gastrostomy, which was performed, under local anesthesia, by Dr. Carl Beck. The patient stood this ordeal very well but could not retain much of the food poured into his stomach; he would vomit or return it through the created gastric fistula. About one week later, while his neck wound was being dressed there came away a large slough which left a perfectly smooth cavity or outline of the thryoid cartilages, with the opening of the esophagus, but no evidence of any carcinoma. Tissues taken at different times from the neck region for microscopic examination showed at no time evidences of carcinoma. It now became possible to insert a stomach tube into this esophageal opening and so feed the patient, allowing the gastric fistula to close. The patient, however, was so markedly weakened that it did no good and he succumbed to the general asthenia. This case demonstrates that the coagulation method destroyed all the carcinoma but at the sacrifice of life. It reminds me of a story which is apropos hence I hope to be pardoned when I insert it in this paper.

"A man that had a cancer on his face was asked by a Patent Medicine Company for a testimonial, because it was known that he was cured by their product. He acquiesced and said: 'After taking ten bottles of your medicine I wish to say that I am glad to state that the cancer of my face is gone, but I am sorry to say also that my face is gone too.'" The loss of this man's larynx may be due to the fact that the principles of Percy's coagulation method were not carried out in the strictest sense, because I had to develop my own technic. I have reference to the cooling of the neighboring tissues which it will be seen was employed in the subsequent cases with not such destructive results.

*Case 2.* Mr. Hgl., 55 years old, married, two healthy children. Brewer by trade, uses his voice often in speech making, and in summer is a great rooter at the baseball games. Lives in Montana in the copper smelting region.

For past six months noted a hoarseness which gradually increased to a marked degree. Consulted his specialist who told him he had a growth in the larynx and removed a piece for microscopical examination. This proved positive for carcinoma. With this history he was referred to me. I found a growth as large as a hazel nut, irregular in outline and situated nearer the left posterior commissure than anteriorly. The physical and laboratory examination, other than the microscopic specimen of the piece removed, were negative. Operation decided upon. Under local anesthesia (morphin ¼ gr. and novocain and cocain) a laryngotomy was performed, the same way as in Case 1, except no preliminary tracheotomy was done, and the cricoid cartilage was opened. The same procedure of removing the growth and cooking its base as in the previous case was done by the Percy method, and no cooling

Fig. 1.

Fig. 2.

device employed. It must be remembered that the growth was located nearer the esophagus than in Case 1, owing to what followed. The thyrotomy was dressed open by packing on top of an up and down tube. The patient made a very rapid recovery but he could not swallow. After the customary forty-eight hours he had to be fed by rectum for more than a week. At the end of that time it was possible to pass a stomach tube and he was fed through that, but still it was impossible for him to swallow, although local conditions appeared very satisfactory. At the end of the third week he could not swallow water but could rice and mush. At the end of five weeks he could swallow anything. The tube was left out and wound permitted to heal.

The patient went back home to Montana, and I heard nothing from him until four months later, when he came in with a tracheotomy tube and marked swelling on the right side of his neck. He stated that he had been feeling

fine, constantly gained in weight, when one day he noted a pain in swallowing, then a sudden appearance of a swelling on the side of his neck, chill and fever and considerable difficulty in breathing. The doctor was called in and hurriedly took him to a hospital where he performed a tracheotomy. This neck growth then disappeared somewhat slowly, but again reappeared, then began to decrease, but now he cannot breath well when the tube is taken out.

Examination shows that the man has not lost any in weight and within the larynx there does not appear to be a recurrence of carcinoma. There was however a sort of an edematous looking tissue about the right cord closing up the larynx. No motion to the arytenoids. The neck mass is not nodular, but smooth, hard and yet appears to fluctuate somewhat. Slight temperature every afternoon. My diagnosis was a possible pharyngo-esophageal diverticulum in consequence of the weakening of the wall, due to the action of the Percy heater. I therefore cut down over the swelling and found a great deal

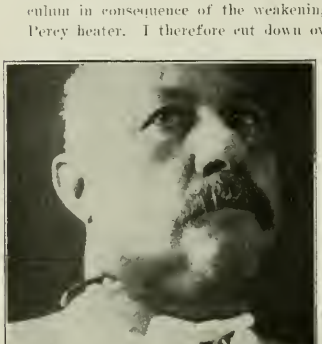

Fig. 3.

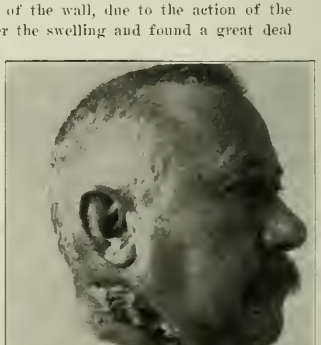

Fig. 4.

of infiltration of the tissues of the neck. I removed some of this infiltrated mass and had it examined microscopically (it was found negative as to cancer). Coming down near the pharyngeal area I opened into a cavity which was filled with a considerable amount of thick fluid containing white particles, which were found microscopically to be necrotic tissue. A drainage tube was inserted and patient observed as to possible recurrence of carcinoma.

Conclusions in this case are:

(1) That he is possibly cured of his carcinoma of the larynx.

(2) That the location of the growth near the esophagus and the removal and subsequent cooking of this carcinomatous base without any cooling apparatus was probably responsible for the weakening of the esophageal wall with subsequent diverticulum formation.

(3) That had the doctor incised the swelling and put in a drain he might have avoided a tracheotomy.

*Subsequent Course.* Within a few days another piece of tissue removed from the neck mass showed a rapidly growing carcinoma. There was very likely a glandular infection present at the time of the first operation, but it could not be determined as the patient had a very short fat neck. I now decided to try out the method of coagulation by the diathermic process which

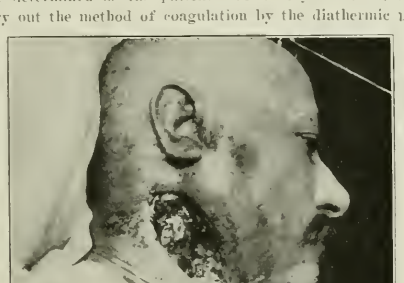

Fig. 5.

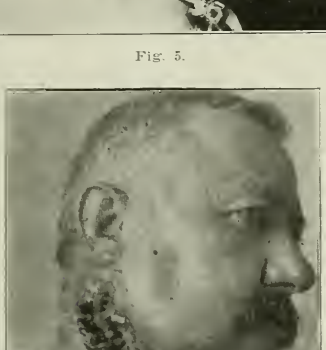

Fig. 6.

I wish now to demonstrate. We inserted into the tumor the flat metal electrode and the sharp needle point electrode opposite this flat surface having about 1 cm. of tumor mass between the two electrodes. Now the current was turned on and from 1,000 to 1,800 milliamperes of electricity sent through. One observed the sizzling of the tissues and if the finger was put on, one would

feel that the tissues were very warm. The measurement of the heat by a thermometer registered 120 Fhr. about one minute after the treatment.

Such applications are made in three to five places at the same seance. Of course the skin and tumor are injected with novocain because the treatment is painful. After six weeks of treatment once or twice a week, we found considerable degree of reduction of the tumor, which is not exactly a necrosis, but a mummification of the mass and falling off with the dressings. There is comparatively little odor to the wound unless neglected to be dressed. It is interesting to note the resistance of nerve and blood vessel to this treatment. The facial nerve is almost exposed and the heat from the treatment must undoubtly reach the nerve, yet the facial muscles are in perfect action. The carotid artery is exposed and fearing possible erosion and fatal hemorrhage, I decided to put a ligature about it at the innominate, thus being able to tie it in event of an accident.

A few days later a sharp oozing occurred, so I decided to ligate the carotid. This however did no good, because the following day a much more severe hemorrhage occurred from which he succumbed.

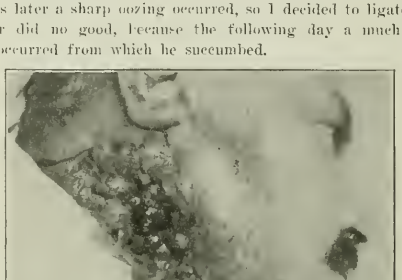

Fig. 7.

*Post mortem* examination of the wound showed that the hemorrhage came from the internal jugular, which I considered thrombosed a long time before this.

The further conclusions in this case are that the carcinoma was not destroyed by the diathermia as we applied it, and that neither nerves or blood vessels are easily affected by the degree of heat obtained by this strong current. The progress of the disease and treatment is illustrated in figures from 1 to 7.

*Case 3.* Mr. Fitch, 51 years. Began to notice a hoarseness in June, 1916. After about three weeks consulted a laryngologist who treated his throat by local applications. On one occasion following a treatment he had a choking spell that alarmed the family. He was also rapidly developing difficulty in breathing, particularly on inspiration. He changed to a laryngologist who recognized a growth and suspected carcinoma.

At this stage the patient was referred to me, exactly seven weeks from the time he started to complain of hoarseness. On examination I found a pale

looking mass the size and shape of a good sized hazel nut, being attached on the right side at and below the vocal cord near the arytenoid region. It was smooth in character and appeared to be quite free and movable, like a polyp. General condition of the patient was excellent. Operation for relief and positive diagnosis, decided upon. This was performed at once under local application to the oral cavity, pharynx and larynx of pure cocain by suspension laryngoscopy.

While doing this the patient gave a sudden marked inspiration and sucked in this pedunculated tumor into the cavity of the larynx, so completely shutting off his air. By means of a long pair of dressing forceps the tumor was brought back outside of the larynx so that the patient could breath again. By means of Killian's alligator forceps the pedicle of this tumor was grasped and without very much effort the whole growth was removed. Very little bleeding followed. The base of this growth remaining in the larynx was now thoroughly curetted and the Percy cautery applied.

Fig. 8.

Fig. 9.

There was very little reaction following this procedure and the patient considered himself cured. The microscopic examination of the tumor removed proved it to be a small spindle celled sarcoma with very few blood vessels and blood lakes. It was decided to do a more radical operation should there be a recurrence. This occurred with such rapidity that within ten days the patient had difficulty in breathing. A laryngotomy by the aid of the Percy (cold iron) coagulation method was performed and the tumor removed. I employed the esophagus cooling tube as well as the Leiters coils externally, thus conforming nearer the true technic of Percy. I closed the laryngeal wound primarily, but left in the tracheotomy tube for a few days. There was considerable toxic absorption from the wound, but the patient made an uneventful recovery and left for his home town three weeks after operation with

the tracheal opening closed. Two weeks later the patient began to show evidences of laryngeal obstruction and the laryngologist in his home city advised him to return to me because he found a rapid recurrence of the growth.

The patient wished to defer doing this a few days, when one day he had a choking spell and the doctor had to do a hurried tracheotomy. As soon as he recovered from the shock, etc., of the last procedure he came back under my care. I found a smooth growth filling up the entire larynx and in the centre a yellowish firm necrotic plug. I decided to reopen the larynx and found the entire ventricle filled out principally with this necrotic plug, which could not be dislodged. The miscroscopic examination of a piece of tissue removed, still showed the sarcoma. It was now decided to give him X-ray treatment by means of deep penetrating rays, right through the larynx fissure which was being held open by a Jackson up and down tube. The patient was now beginning to show marked symptoms of toxic absorption

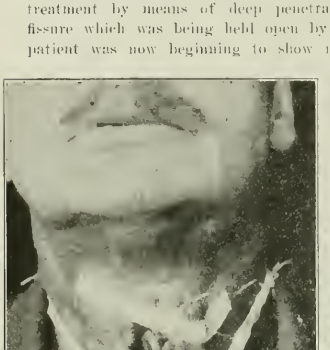

Fig. 10.

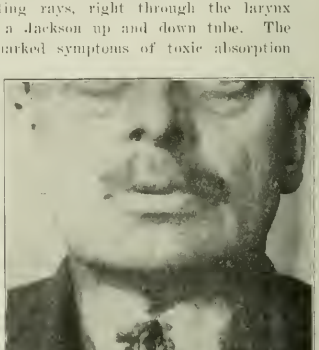

Fig. 11.

and it was thought best to discontinue the X-rays and try massive doses of radium. Therefore in January, 1917, the various shaped tubes and applicators of radium were placed within and without the larynx and allowed to remain 12 hours at a time, for thirty-six hours. Altogether 200 mlgrs. of pure radium were used at each sitting. The immediate result, that is within a day or two was striking. The odor and what little pain there was, became much less. On the sixth day we could all see a decided shrinking of the sarcomatous mass, but the existing necrotic plug from the Percy treatment did not appear to be influenced, except perhaps as to the toxic absorption from it being less.

*Subsequent Course.* During the next three weeks the sarcomatous mass was seen to be shrinking, the neighboring skin of the neck much reddened and tender to superficial touch. Even the necrotic plug became loosened up, but whether that was due to the action of the radium or was about due to come off is problematical. The fact remains that thus far three weeks after the

radium has been applied, he is in a better condition than he has been before the first operation. He is gaining in weight and is very hopeful of a cure. It is of course, my intention to make a subsequent report of this case.

The progress of the disease and treatment up to date of this paper are shown in figures from 8 to 11.

(Note). Since the presentation of this paper in February, 1917, the patient shortly after began to show evidences of marked toxic absorption, also rapid emaciation and evidences of increase in the necrotic mass. He began to have great difficulty in swallowing with marked infiltration of the esophagotracheal region, which lead to a speedy end.

The final conclusions in this case are that neither the operation, Percy cautery, nor the X-rays and the radium were able to stop the growth of sarcoma. While the necrotic mass following the application of the radium and the result of the original burning with the Percy cautery showed evidences of destruction, there appeared to be definitely outlined a new growth of sarcomatous tissue. Another important point is that in the future when I make a diagnosis of this condition of sarcoma of the larynx, I shall insist that a laryngectomy be the only procedure warranted.

*Case 4.* Mr. M. This case has already been reported in the Annals of Otology, Rhinology and Laryngology, (March, 1917), as a probably cured case of carcinoma of the larynx by means of operation by suspension and deep penetrating X-ray treatments. I will, however, make an abstract of that report in this connection, since that will save time and be more complete.

Patient age 54, always in good health, slight hoarseness of three years standing. Consulted laryngologist who found a small node anteriorly on left cord. Removed a particle, which was sectioned and diagnosed by competent pathologists as squamous celled carcinoma. This the patient brought to me with the suggestion from his laryngologist to remove the larynx if it was the only thing to do, but try and save the voice if possible. The laryngoscopic examination verified the previously stated picture and it was decided to do a thyrotomy but to subject him to a preliminary deep X-ray treatment to possibly prevent implantation and sealing up of the lymphatics. The result from the X-rays (ten treatments) was remarkable in the reduction of the swelling and clearing of the voice. I now decided to change my plan and therefore suspended him and removed all the growth I could find. This proved to be microscopically, carcinomatous. I had him X-rayed again for three erythema doses, twenty-six treatments in all, in the hope of completely eradicating the disease. He did beautifully in so far as the action and appearance of the larynx. There developed, however, a complication from the X-ray treatment, that was somewhat alarming, namely symptoms of myxedema. These promptly disappeared on the administration of thyroid gland. Patient went back to the coast apparently a cured man. After nearly a year he returned saying that the doctor thought there was a recurrence, and that his voice was not clear. Examination verified these facts. I again suspended him, removed some small particles from the left vocal cord, anteriorly, which showed microscopically not to be malignant. He received, nevertheless another course of X-ray treatment and went back home apparently cured. Almost another year passed when he wrote saying there was a return of the trouble and I advised him to come back. Examination now revealed a considerable swelling at the seat of the old trouble,

but it appeared to be smooth. I again suspended him and removed some of this swelling and subjected it to the microscope. Again report normal cells, principally inflammatory. I now treated him locally, with astringents, but instead of getting smaller, it grew somewhat rapidly and looked cancerous. After a number of X-ray treatments were given and no improvement seen, it was decided to operate more radically. Since the results from the thyrotomy with subsequent Percy coagulation treatment proved not to be successful in my hands, I proposed a laryngectomy, which the patient accepted, reluctantly, on account of the prospects of losing his voice. After promising and describing to him my idea of an artificial larynx, and the good prognosis for life if he was operated before the cancer developed outside of the larynx, the patient consented. Under local anesthesia, apothesin one half per cent. solution and novocain one per cent. solution, the following technic was employed:

Fig. 12.

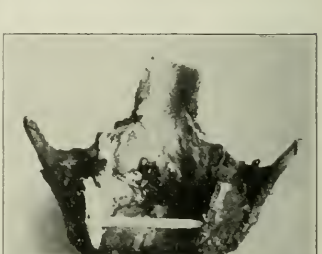

Fig. 13.

A T shaped incision was made, the horizontal incision passing above the hyoid bone and the vertical from the middle of the horizontal incision down to within an inch of the sternal notch. Severing the sternohyoid the sternothyroid muscles about their middle, dissecting freely the trachea from the two lobes of the thyroid gland, I packed strips of gauze all around so as to wall off the mediastinum. After very carefully dissecting the trachea from the anterior wall of the esophagus, I severed it (the trachea) from the larynx and turned it down toward the sternal notch allowing it to protrude somewhat. It was stitched to the skin on either side. The larynx was now dissected upwards away from the esophagus until the pharynx was reached. Then the thyrohyoid membrane was severed and the larynx removed, closed, with sufficiently healthy tissue to insure that none of the malignant growth was left behind. The defect in the pharynx was reduced to the minimum and the

incision united except in the centre just below the hyoid bone which is to become the subsequent place for the insertion of the artificial larynx. Considerable catgut was employed in the obliteration of the cavity. This became infected and there resulted a great deal of sloughing. In order to keep the food from coming out through the pharyngeal opening it was necessary to feed the patient with a stomach tube. Gradually as this opening contracted, the patient could swallow without difficulty. Outside of this trouble, an uneventful recovery resulted, as shown in Fig. 12.

The gross specimen, Figure 13, when open showed that the carcinomatous process was confined entirely within the larynx, but markedly involved the base of the epiglottis. A microscopical examination of this growth from the depths shows positive carcinoma. The next step was to construct an artifical larynx. The development and evolution of it would be very interesting to narrate, but since the same is not yet perfected, I will defer the detail description

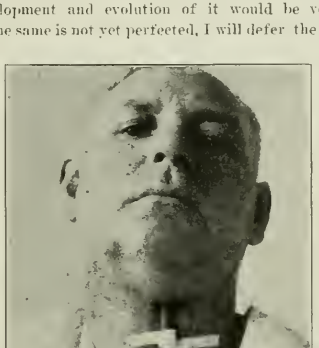

Fig. 14.

for some future publication. Suffice it to say that a fairly stiff rubber tube was found to be the best material. It is about 2½ inches long by ½ inch in diameter. The upper end opening has a sort of a nipple in order to confine it to the opening in the pharynx, while the lower tracheal end is smooth and can move up and down according to the motion of the head and neck. It, however fits fairly tight, so as not to permit any escape of air while speaking. By means of this simple device (Fig. 14), the patient can whisper very well and can be clearly understood. Since it is covered by the patient wearing a handkerchief or band about the neck, most people think that the patient has a cold. The method of producing tone is now being experimented with on this patient. I have applied a means of compressing this rubber tube from the outside, thus narrowing its caliber at one place, making a sort of a chink and he is able to produce a sound. A couple of elastic bands worked right into the rubber tube as had been suggested to me by Dr. Iglauer, is also a

possibility. Whenever the patient eats, he is compelled to remove this speaking device, but I believe that that objection will also be overcome in time, in that the patient will be able to get the head in such a position that nothing will pass into the tube. He is able to do it now at times.

In conclusion, I wish to say that this patient has all the rights to expect a complete cure from cancer, but a subsequent report will be necessary. If when this patient succumbs from no matter what cause, outside of cancer, he is not thoroughly examined post mortemally, we will not know whether he was cured or not.

## DISCUSSION.

**Dr. T. J. Harris,** New York: Just a single word of commendation for the work which Dr. Beck has been doing. He deserves the heartiest commendation for his perseverance and for the amount of research work in all directions which he has carried out. It is my sincere hope that out of it all will come something that is worth while. Much of what he said is new to me. There are one or two points. One is as to the result of Dr. Coakley's work in conjunction with Dr. Burnham of Johns Hopkins in the use of radium in carcinoma of the larynx. The radium was applied externally. He showed three cases last year at the New York Academy of Medicine, and those cases could certainly be regarded as arrested cases. The growth had disappeared. He took the same view of the matter that any of us would take, that it was too early to predict a permanent cure. His only fear was that the growth would return. It is very interesting to know, however, that radium applied externally would be able to cause the disappearance of the growth. My own experience has been absolutely futile as regards the use of radium in carcinoma of the esophagus, or larynx, applied internally.

The second point is in regard to the ingenious method of artificial voice. Some of you perhaps remember the celebrated case of Dr. Cohen, in which the man had a natural voice, presumably pharyngeal, which could be understood across a room. Many methods have been devised, but none, so far as I know, has been regarded as successful. If what Dr. Beck has is better it will be a great addition to these cases.

I am interested in having Dr. Beck tell in conclusion what emphasis he puts in a broad way on the operative treatment of carcinoma of the larynx. Dr. Mackenty of the Manhattan has been getting excellent results. He has reported, and will possibly again report his cases so far as his results go.

**Dr. Samuel Iglauer,** Cincinnati: I would like to ask Dr. Beck if he has tried intubation with radium? I have had one or two cases where it was possible to intubate the patient, using an ordinary intubation tube, and fastening the radium capsule on the outside of the tube with adhesive plaster. The patient can then breathe in a natural way while receiving an internal application of radium.

**Dr. Harris:** What are your results?

**Dr. Iglauer:** I was not discussing results, but there was no permanent benefit. The point I wished to emphasize was that the ordinary method of treating the larynx with radium requires tracheotomy, while this method obviates tracheotomy.

**Dr. Beck** (closing the discussion): answering Dr. Iglauer: I have not employed intubation for the application of radium. Most cases have been very far advanced, and tracheotomy was the first thing I had to do. However, in this one case in which I was banking on X-ray to

do the work, that probably would have been the method I would have employed, but the quantity of radium, as stated, which is necessary in these cases, would scarcely go into the larynx and the patient still be able to breathe. The 125 milligrams of radium which I am using in these cases I have in four separate tubes, one 50 and three 25 milligrams. That is the way we have them, and they have to be put into the larynx and about it. We have the Rogers' arrangement, the tracheotomy and intubation tubes combined. We take the regular radium capsule out of the container and put it into this tube made for it within the intubation tube.

**Dr. Loeb:** I would like the doctor to explain why he has given up thyroidotomy?

**Dr. Beck:** From the fact that in every case but two in which I have tried it there has been recurrence. I so thoroughly believe in Dr. Crile's work, and his idea in regard to the implantation, that I think the moment you cut into it, even in doing a tracheotomy for that matter, there is danger of cancer implantation. Bloodgood has said, that on operable carcinoma anywhere in the body, that is, within the areas that may be removed—to open into a carcinoma like that is as much to be condemned as is the general practitioner who procrastinates in examining for that condition. Do not open into a cancer of the larynx if you expect to get a cure.

**Dr. Harris:** Just a further word. What I thought was the most encouraging part of the report, was that of the deep penetration in the treatment of sarcoma.

**Dr. Beck:** In the pharynx cases you get the most wonderful immediate effect. In some cases there is complete disappearance. If I may take two or three minutes to describe a case of retromaxillary sarcoma. The man had a sinking in of the eye, paralysis of the tongue, paralysis of the palate, paralysis of the larynx, on one side complete blockage of the nose; intense pain, so that even large doses of morphin would not satisfy. That patient had three erythema doses of deep penetration, X-ray treatment, and that required about thirty seances. Every one of these symptoms have disappeared. The diagnosis in this case was cinched by the microscope as a spindle celled sarcoma.

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NOTE.—Since reading this paper and reporting the marvelous effect of deep penetrating x-rays on this as well as other cases of sarcoma, there occurred a rapid recurrence with very sudden termination of the lives of these patients. In one word the more rapidly the sarcoma disappears following the use of x-ray and radium the more rapid is its recurrence and fatal termination.

# REPORT OF A CASE OF BILATERAL, CONGENITAL, OSSEOUS ATRESIA OF THE EXTERNAL AUDITORY CANAL WITH AN EXCEPTIONALLY GOOD FUNCTIONAL RESULT FOLLOWING OPERATION.

By L. W. DEAN, M. D. and T. R. GITTINS, M. D., Iowa City, Iowa.

*Occurrence.* According to Bezold and Siebenmann[1] this condition occurs once in every 2000 cases, and one out of every six cases is bilateral.

In 1909, Forsyth[2] makes the statement that but two cases had been reported of bilateral atresia in which there was hearing and understanding of speech.

T. E. Francis[3] in 1910 speaks of one case of unilateral osseous atresia in 10,000 cases examined.

Pomeroy[4] in his book "Diseases of the Ear," 1886, makes no mention of congenital osseous atresia of the ear canal. Likewise, Politzer's[5] work, translated by Sir Wm. Dalby in 1894 speaks only of acquired atresia, not even noting a congenital form.

*Pathology.* The pathological picture of a typical case of congenital osseous atresia as described by Bezold and Siebenmann[1] is as follows:

"A small irregularly formed rudiment of cartilage which is covered with skin is on the outside in place of the auricle. There is no entrance into the meatus. The mastoid process usually starts directly behind the glenoid process of the lower jaw. Usually, there are rudiments of two ossicles and no drumhead is present. The antrum of the mastoid is most often quite well developed."

These writers give an account of the work of Joel[6] in 1894 at which time he compiled records from cases at postmortem. He found aplasia of the tympanum and also the drum-membrane. The labyrinth and its windows, together with the stapes were nearly always found intact. In almost all of the cases, the osseous portion of the canal was represented by a mass of compact or cancellous tissue; occasionally a small defect in the bone could be observed, or occasionally a narrow fissure leading to the tympanum. Usually the tympanum was narrowed or replaced while the Eustachian tube was sometimes abnormally wide.

There has been much discussion as to the probable pathology in atresia. Some writers basing their arguments on clinical observations, others on their studies of the embryological development of the ear. One group claims that when the meatus is deformed, that is, congenitally occluded with bone, the internal ear is also affected; the other group holding that the external ear can be and usually is deformed without the inner ear being affected.

The work by Page[7] in 1914 presents the most complete review of this subject.

Giving Politzer and Kerrison as his authorities, he says that the bony meatus is not associated with the development of the essential organ of hearing. The greater part of the tympanum itself, including the osseous portion of the Eustachian tube, except a small part of the anterior tympanic wall and roof, develops with the essential organ of hearing in the petromastoid portion, and is not associated with the development of the meatus and auricle in any way. He goes on to say that according to Politzer, the stapes develops from the labyrinthine capsule and a ring-shaped deposit situated laterally to the labyrinthine capsule, thus explaining the fact that the stapes is rarely found missing in these atresia cases while the other ossicles may be.

On the authority of Cassels,[8] writing in the Glasgow Medical Journal in 1876 Page says: "Even in cases where there is total absence of the Eustachian tube accompanied by absence of an external auditory meatus and tympanic cavity, a normal labyrinth will usually be found, and in all probability a stapes."

Quoting Alexander[9] (Zeitschrift fur Ohrenheilkunde 1908) he says: Conclusions were reached after many autopsies by this writer that in the vast majority of cases a deformity of the ear was limited to either the conducting apparatus or the perceiving apparatus, alone.

Page[7] also quotes Costex and Berruyer who in turn accredit Alexander with the observation that a malformation of the labyrinth always affects the facial nerve and auditory at the same time, and that one may conclude from this that all malformations of the external ear coexistent with facial paralysis indicate alteration in the labyrinth.

In concluding his discussion Page[7] gives as his own opinion that it can definitely be stated that cases of defective development of the internal ear are rarely, if ever, found associated with atresia of the external ear in otherwise well-developed infants.

The development of the ear as given by Doctor Prentiss, Head of the Department of Anatomy, University of Iowa, agrees with the above conclusions. He describes the middle ear as an uppushing of the first visceral cleft, the malleus and incus as formed from the upper extremity of Meckel's cartilage. The external auditory canal and drumhead are evidence of the inpushing of ectoderm from without, the uppushing of entoderm from within and the overgrowth outward of the skull to accommodate the growth of the brain. The internal ear is described as derived from the otic vesicle, which pushes in from the ectoderm, then becomes entirely separated from the exterior.

The following represents opinions contrary to these just expressed:

1. Kopetzky[10] in 1908 makes the statement that the graver forms of atresia are usually associated with maldevelopment of the middle and internal ears, it being rare to find a well-developed concha. He gives as his authority Link, Jacobson, Blau, L. Wolff and Brieger. He also quotes Schwartze as saying that invariably with grave one-sided atresia, a maldeveloped labyrinth also exists on the affected side, and substantiates his observations by the fact that a test fork on the the middle of the top of the head is heard only in the normal ear.

2. Forsyth, F. A.[2] in 1909 states, that congenital atresia is usually associated with malformation of the auricle and in most cases with malformation of the middle and internal ear.

*Prognosis.* In going over the literature on this subject, one is forcibly impressed that the general trend of opinion of the best men has been and still is against operative interference.

Dench[11] in 1900 discourages any operative work in osseous atresia cases, saying that results do not warrant attempt at interference.

Hovell, T. Mark[12] in 1894, says that prognosis is bad because of the chance of trouble in the inner ear. He advises against interference in unilateral cases and considers no operation is practicable in cases where an osseous mass occupies the place of the external auditory meatus.

Kopetzky[10] states that results of operation are usually bad if functionating apparatus seems to be at all affected.

Phillips, W. A.[13] in 1891 gives as his opinion that congenital atresia of the external auditory canal should be let alone because of the bad results.

Beck, J. C.[14] says not to operate for the same reason.

Ballenger[15] advises against operative interference in these cases.

Andrews[16] gives a fair prognosis based on a case in which mastoid route was taken and hearing was somewhat improved.

Hershel[17] says that prognosis is very bad, basing his opinion on one case of his own and four cases which he found in the literature.

Bishop[18] in 1890 in discussing imperforate auditory canals where there is osseous occlusion advises against operation and states that the best authorities were at this time against operative interference unless there was only a thin bony septum. He mentions that Politzer advised against operation unless this condition was present.

Milligan[19] in 1910 says only to operate when one could palpate the annulus and could demonstrate a passage of air to the middle ear through the Eustachian tube.

Page[7] in his work of 1914 is practically the only writer who gives an at all favorable prognosis in these osseous atresia cases. He says that "operation is warranted in practically every case of bilateral congenital microtia with total osseous atresia which occurs in an otherwise well-formed infant." In his writing he gives the following as the most prevalent reasons given as a basis of non-interference:

1. That where the external auditory canal is deformed and the meatus congenitally occluded, there invariably exists such extensive defect either in the conducting mechanism, or in the perceptive apparatus, or both, that operation is unwarranted.

As an argument against this theory, he refers to the developmental facts and postmortem findings referred to in this paper under the discussion of the pathology.

2. That the operation should not be performed unless tuning forks and bone conduction tests are found to indicate a functionating mechanism.

3. That it is practically impossible to prevent closure of an artificial canal after it has been made.

This last reason he disposes of by referring to the method of doing a mastoidectomy, then skin-grafting the cavity as was done in his case.

*Treatment.* The following operations I have found described:

1. Bezold and Siebenmann[1] in 1908 describe an operation suggested and tried by Schriebe.

This operator opened into the antrum from the mastoid, made a wide canal by lining the mastoid cavity with Thiersch grafts.

2. Kopetzky[10] in 1908 cites an operation of Jaeger in which the mastoid was opened down to the antrum and then left open posteriorly, thus allowing sound waves to reach the middle ear through the mastoid cavity and aditus.

3. Andrews[16] gives record of a case operated upon by the mastoid route. Skin flaps were made and turned down into the wound.

4. Hershel[17] suggests a radical mastoid operation, but indicates a bad prognosis.

5. Dench[11] in 1900 describes an operation in which the canal is made in the usual site, then kept open by means of a rubber or metal tube until cicatrization takes place.

6. Alexander[9] in 1908 gives history of one case in which a mastoidectomy was done, and skin flaps were made from the posterior part of the wound. The meatus thus formed was packed.

7. Duel[20] describes a case in which both mastoids were opened and the cells cleaned out. Skin grafts were used in the wounds to form the meatus, but without result as the wounds were closed with no improvement in hearing.

8. Beren's[21] case. This case operated by Berens was described by Duel. A radical mastod operation was done on each side, then the cavity was dermatized with flaps.

9. Page.[7] The mastoid cavity was opened, and the cells cleaned out down to the tympanum. An opening was made in the auricle, then the mastoid incision was closed. Ten days later a skin graft in the mastoid wound, through the artificial meatus was done.

## REPORT OF CASES.

1. Alexander's case (9) Zur Chirurgischen Behandlung der kongenitalen. Zeitschrift fur Ohrenheilkunde 55:144 (1908). Reported by Page (7).

This was a case of a boy eleven years of age, with a normal left ear and congenital microtia with total bony atresia in the right. Operation was performed for mastoiditis.

He found the absence of a bony canal, pus in the mastoid cells and ossicles present. The drumhead was absent, a bony plate being in its place.

The operation was completed by tucking in skin flaps from the posterior part of the wound. The meatus thus formed was packed. Eight months after the operation, the meatus was 4 mm. wide.

As a result he gives an improvement in hearing from 1 metre through a tube before operation, to 4 or 5 metres after operation.

2. Blumenthal's case (22). Mention was made of two cases in which results of operation were very unsatisfactory. This writer argues against operation, saying that one should keep what hearing was already present.

3. Duel's case (20). This case was a girl twelve years of age with a bilateral total osseous occlusion of the ear canals.

Both mastoids were operated, then skin-grafts were applied in the wounds. The wounds failed to keep open and there was no improvement in hearing.

4. Dahmer's case (23). In this instance the patient was three years old and had a bilateral osseous atresia.

Operation was performed on each side, a bony canal being made to the tympanum. Drumheads were not present. The stapes were present on each side.

The author reports that the hearing was much improved.

5. Morris' case (24). This was a case of a boy ten years of age with a bilateral bony atresia. The patient could hear loud voice. The mastoids were opened. The author does not give results nor describe operation.

6. Braun's case (7). Page reports this case, saying it was unpublished at the time of his work.

The findings at operation were that the jaw encroached on the line of the canal, annulus was present, but no drumhead was found. Ossicles were found in a practically normal tympanum.

The author gives as a result a marked improvement in hearing.

7. Cheatles' case (25). This author reports a case of congenital malformation of the auricle and meatus with unilateral osseous atresia. There was a fairly well formed auricle without a meatal opening. Some hearing was present and vestibular reactions were positive. The X ray picture showed mastoid cells and middle ear tract. The jaw appeared to articulate with the posterior meatal wall.

At the time of the operation no mastoid cells and no tympanic plate were found. After finding no meatus, the operator stopped.

8. Beren's case (7). This case was reported by Page, who said that up to the time of his work it had not been reported.

The patient was five years of age. He had a bilateral osseous atresia of the ear canals. Both sides showed articulation of the jaw with the anterior wall of the mastoid.

A radical mastoid operation was done on each side. No drumheads were found. On one side a rudimentary malleus and incus were found, on the other a rudimentary malleus, while the stapes was present on each side.

The mastoid cavity was dermatized with skin flaps.

A good result was reported.

9. Hershel's case (17). In 1909 this writer reports a case of congenital atresia of the auditory meatus in which the auricle was normally developed.

The patient, an old lady, was the offspring of blood-relations. The hearing was 10 cms. for whispered voice.

The meatus was found occluded by a plate of bone. The Eustachian tube was found to be patent.

On postmortem (no operation was performed) no drum membrane was present.

10. Page's case (7). The patient was two years of age with a bilateral osseous atresia.

One ear was operated at this time. The mastoid was opened (mucopurulent material found). The antrum was located, small aditus and contracted middle ear were exposed.

There was no fibrous canal, drumhead or annulus found, likewise no ossicles. The eustachian tube was open.

An opening was made in the auricle, then the posterior incision was closed. After ten days' time, a skin graft was done. Caloric test was positive.

Two years later the meatus was 5 mm. in diameter, the cavity was clean and the hearing was improved.

Later the other ear was operated. No meatus or bony canal was found, likewise no ossicles. An indefinite drumhead and poorly-formed malleus were present, while the incus was quite well formed. The stapes was present as was afterwards demonstrated.

An artificial meatus was formed, then Thiersch grafts were applied ten days later.

Caloric test was positive.

Three years after the first operation the mother of the patient says child can hear natural tone of voice.

After a study of his case and those reported in the literature, Page (7) makes the following conclusions:

1. Operation is warranted in practically every case of bilateral congenital microtia with total osseous atresia which occurs in an otherwise well-formed infant.

2. After a child with this deformity has reached two years of age, X-rays should be taken, then the mastoid opened. If a drumhead is not found, the contents of the tympanum with the exception of the stapes should be removed.

3. Injury to the jaw can be avoided by keeping well posteriorly.

4. It is best to allow a large cavity, then line it with skin grafts.

5. The operation should be done early to allow the development of speech.

Patient, L. McC. Male. Age, 15. White.

E. C. poor hearing.

The patient came to the University Hospital, June 21, 1916, complaining of poor hearing.

The patient's hearing had been poor since birth. When two years of age he began learning to talk.

The patient says his hearing has been failing more rapidly during the past year. There is some tinnitus at times. He says he hears best when on a train or anywhere where there are loud noises. There has been some dizziness at times, never marked. At one time such a dizzy spell was associated with staggering and vomiting. This was about six years ago.

The patient is in the seventh grade, has always seemed quite backward. Parents think he does not talk as plainly as he should.

The child was born at full-term after a normal labor. He is the third child in a family of nine. There are no other deformities in the family. There are no children dead. There is no history of miscarriages. No history of consanguinity can be obtained. Patient has had measles and mumps; has not had scarlet fever or diphtheria.

*Examination.* Ears.

Right ear. Auricle appears normal in size and position. Where the external meatus should be is a concavity 3 mm. deep. Probing shows it to be bone covered with skin.

Hearing with alarm. Forced whispered voice, three inches. Spoken voice, five feet.

Hearing without alarm. Same.

Rinné. Negative. Bone conduction slightly less than normal. Air conductions much cut down.

Weber's test. Localizes to the left.

Low notes are markedly cut down. Hears C' fork only at marked intensity.

High notes are cut down, but are heard better than lower ones. Hears C4 fork at high intensity.

Left ear. The left ear is similar to the right except that the external auditory canal extends inward about $\frac{1}{2}$ cm. Probe again comes against epidermis covering over what is apparently solid bone.

Hearing with alarm. Forced whispered voice, two inches. Spoken voice, three feet.

*Vestibular test.*

1. Rotation test. Patient was rotated from left to right ten turns in twenty seconds. Nystagmus, almost wholly horizontal in type, was present on looking to the left, and lasted thirty-five seconds.

After rotating from right to left, nystagmus was present on looking to the right for forty seconds. Patient became quite nauseated and vomited profusely ten minutes after completion of the rotation tests.

2. Caloric test. Water at 68 degrees was applied to each remnant of ear canal for six minutes. No nystagmus, vertigo or nausea resulted. Ice water was applied in the same way for six minutes. No nystagmus, vertigo or nausea resulted.

Nasopharynx. Orifice of each Eustachian tube appeared smaller than normal, but appeared to be patent. The tissues surrounding the orifice appeared to be atrophied.

Wassermann on blood is negative.

X rays of mastoids. Shows mastoid cells on both sides apparently normal. An area is made out in each plate which is apparently tympanic cavity.

On June 26, 1916, the left ear was operated upon with the hope of establishing a meatus, the supposition being that the perceiving apparatus was intact and functionating.

*Operation.* The left mastoid was opened and a simple mastoidectomy was done in the usual manner. Cells seemed to be normal. The antrum of the mastoid was found, aditus was approached and enlarged until the incus was well exposed. The incus appeared normal and movable.

Taking the floor of the aditus as a guide for the facial, the bone external to the tympanic cavity was attacked. Solid bone about one-third the thickness of a normal canal was found between the mastoid cells and glenoid fossa. This was removed with the burr working outwards to the concave area representing the external meatus. No annulus could be detected. Just external to the proper position for the annulus, a small cavity 3 mm. in diameter was found. This communicated with the tympanic cavity by means of a very small opening into which a mastoid seeker fitted tightly. No evidence of a drumhead was to be seen. A seeker introduced from without into this aperture caused a movement of the incus. The external wall of the tympanum was so constricted in the anteroposterior direction that it was not considered advisable to attempt the enlargement of this opening. The solid bone was removed externally to the concave depression in the concha. A large opening was made in the concha. The mastoid cavity was lined with Thiersch grafts, and the wound closed posteriorly. The grafts were held with

the usual cotton pledgets dipped in aristole powder. The last of the packing being done through the opening in the concha.

This operation resembled a meato-mastoid operation with primary skin-graft.

On the third day following the operation the cotton pledgets were removed. There was practically no odor, graft seemed to be holding perfectly. There was no pus and a very small amount of discharge. A light iodosyl drain was put in the wound very loosely. Facial, O. K.

July 1, 1916. Graft holding very well. Practically no odor. Dressing just moistened with discharge. Hearing without alarm. Whispered voice, twelve feet. Spoken voice, forty-five feet.

July 6, 1916. Graft has held best in the region of the aditus. There has been a small amount of discharge each day. Apparently no pus.

July 16, 1916. Most of the superficial epidermis of the graft has sloughed. Holding well in the region of the aditus, slight bulging posteriorly. Pack of loose gauze saturated with discharge. No bad odor. Hearing with alarm. Whispered voice, three feet. Spoken voice, nine feet. Hearing without alarm. Whispered voice, ten feet. Spoken voice, forty feet.

July 21, 1916. Graft continues to look fine. There is quite a free discharge. The hearing is the same as on July 16, 1916.

August 10, 1916. The wound is covered with healthy looking epidermis except in one small area posteriorly. The hearing is the same as on July 16, 1916.

August 26, 1916. The wound has been gradually healing and the discharge has almost ceased. There is a smooth epidermal layer over the region of the aditus.

Hearing with alarm. Whispered voice, three feet. Spoken voice, ten feet.

Hearing without alarm. Whispered voice, twelve feet. Spoken voice, forty-eight feet.

The canal is about 1 cm. in diameter at the smallest place.

September 3, 1916. The right ear was operated.

A simple mastoidectomy was performed. All of the cells were removed. The antrum was entered, and the aditus exposed. The horizontal semicircular canal and long process of the incus were exposed. The incus was movable, but was not nearly so free as it should be. It seemed somewhat deformed and fixed at the lower joint.

No trace of any cavity external to the position of the annulus on this side was discovered. A thick wall of bone between the mastoid cells and the glenoid fossa was reamed out using the burr. The soft tissues of the fossa were accidently exposed. From this there was no disturbance in the temporomandibular articulation at any time. No annulus or drumhead was seen. The bone external to the tympanum was not removed.

The lateral sinus and dura in the middle fossa were exposed and so primary skin-grafting was not attempted.

Just internal to the concave depression representing to us the position of the external auditory meatus was a little cartilage. This did not penetrate the bone, but fused with the tissues of the glenoid fossa.

An opening was made in the concha, the posterior wound closed, and the mastoid packed through the opening in the concha.

The facial was O. K. after the patient regained consciousness.

September 7, 1916. The dressings were removed. Hearing with alarm. Right ear. Whispered voice, two feet. Spoken voice, eight feet. Hearing without alarm. Left ear. Whispered voice, fifteen feet. Spoken voice, forty-eight feet.

September 15, 1916. The wound is granulating nicely. Hearing with alarm. Right ear. Whispered voice, two and one-half feet. Spoken voice, nine feet. Hearing without alarm. Left ear. Whispered voice, eighteen feet. Spoken voice, fifty-four feet.

September 20, 1916. The wound on the right side is still open posteriorly. This is being packed daily. A light pack is placed in the left ear each day. A small area near the external meatus has not epidermized as yet.

September 22, 1916. The wound on the right side was grafted today. The original mastoid incision was opened throughout its extent again, exuberant granulations were curetted, Thiersch graft from the leg was placed in the wound, covering over smoothly the region of the aditus. The wound was closed posteriorly and cotton pledgets packed in through the meatus to hold the graft in position. The facial was O. K. after the patient regained consciousness.

September 26, 1916. The cotton pledgets were removed. The graft appears clean and white. A plain linen pack was put in loosely. No bad odor. There is quite a free discharge.

October 1, 1916. The dressings in each ear are being changed daily. The graft in the right ear is holding very well. There is a small amount of discharge on the pack each day. The meatus in each ear is about one cm. in diameter.

October 5, 1916. On this date the left ear which was operated first presents a meatal opening of from eight to ten mm. in diameter. The canal from the meatus down to the region of the aditus is considerably larger in diameter and covered with healthy looking epidermis except for one small area posteriorly. The graft in the right ear is holding very well. There is less discharge each day and the meatus is about ten mm. in diameter.

A review of the literature has brought out no case with such improvement in hearing and satisfactory meatal opening and canal as has this case. The case reported by Page is the best result so far reported and the improvement in this instance in the hearing was not accurately determined so that no definite statement could be made.

The findings on February 1, 1917. Seven months after the operation on the left mastoid the hearing in the left ear was: Whispered voice, forty feet, without the alarm. The right ear was not tested because of an acute middle ear otitis which was present at this time. A few days before the membrane covering the aditus bulged and ruptured. Now there is a suppurative discharge from its perforation.

Examination of the left ear. The external meatus is 1 cm. in diameter. The mastoid cavity is completely covered with white dry skin. There is nothing that looks like an external canal. At the bottom of this cavity the aditus cannot be seen. The site of the aditus is a perfectly smooth concave wall. The small space external to the annulus mentioned in the early description has been filled in and disappeared. The external wall of the tympanum cannot be seen as such. The aditus is closed over by what seems

to be a tough fibrous membrane which is undoubtedly acting as a tympanic membrane.

Examination of the right ear. Four weeks ago this ear was seen. At that time the interior of the ear was the same as the left. The hearing in the right ear was not so good as the left.

A peculiar thing in connection with this case in testing the hearing has always been the way in which the hearing was cut down by using the alarm apparatus. This has always interfered with the testing of the exact amount of hearing in the poor ear.

About two weeks ago the patient complained of an earache in the right ear. On examination this fibrous membrane and skin covering the aditus in the right ear was found to be bulging. The next day the bulging skin ruptured, and up to the time of examination, February 2, there has been a suppurative discharge from the tympanum. The membrane covering it is perforated and looks very much like a perforated normal tympanum.

Our expectation is that with treatment of the suppurative middle ear otitis this membrane covering the aditus will heal the same as the tympanic membrane does.

At this time the discharge from the right ear has ceased and the membrane covering the aditus has healed in a manner similar to the healing of a perforated drumhead.

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## DISCUSSION.

**Dr. Norval H. Pierce,** Chicago, Ill.: I have not had very much experience in these cases. I think a review of the subject would lead one to believe that this external deformity is very frequently associated with deformity of the sound-conducting apparatus, that is, the deformities of the internal ear. It is very difficult to make tests in these cases in young children—to find out by functional tests whether there is a sound preserving apparatus or not, or whether it is deformed or not. In later life I think it is pretty well established, that is, after puberty where the patients can give intelligent answers, that there is more or less, usually more, defect of the sound preserving apparatus.

I have operated on these cases with not nearly the good results which Dr. Dean reports, largely owing to the fact that I find it very difficult to keep an opening into the antrum. Of course that is the universally recognized method of operating, get into the antrum and establish a canal irrespective of where it should naturally be—but establish a canal which keeps an opening into the antrum.

I have been discouraged also by results in operations for microtia, or defects in the conformation of the external ear. Most of these are little or no better after the best efforts that I am able to put forth. Still this paper of Dr. Dean's draws attention to the fact that we can, by properly operating, greatly increase the hearing power of some of these little children.

I think the main thing is to establish—and this will be for the research of the future to establish—what we may expect as to the sound preserving apparatus, whether in existence at the time of the operation or not. On that, of course, depends the prognosis absolutely. If the sound preserving apparatus is present, then in all probability there may be a deformed conducting apparatus. If that is present, and if we can establish a canal leading to the antrum, which will keep open, then the hearing will be benefited.

**Dr. J. W. Murphy,** Cincinnati, Ohio: The report of this case of Dr. Dean's is very interesting. I would like to report in this connection a case which I now have under observation. The child is now four years old. I have had it under observation since birth. I first saw it when it was three months old. The child is a monstrosity, the head, face, and everything being badly deformed. There are no external ears, but only a little portion of cartilage in place of where the auricle should be. I told the parents I would do nothing with the child until it was a little older, and that we would then see if anything could be done in the way of finding an external auditory canal, knowing the difficulty in operations of this kind. The case passed from my observation for about four years. They returned last year, with a request that the tonsils and adenoids be removed. I then discovered that they had been pretty well over the country with the child, seeking relief. They had been to Rochester where the Mayos had operated both ears. There were scars back of the ears showing where the incisions had been made, but I do not think they went into the bone. I could find no depression. The father reported that the operation was negative so far as finding any external auditory canal. In operating on the child for adenoids I examined the Eustachian tubes, and they seemed

about normal. I was going to see what a bougie would do, but the child did badly under the anesthetic, and I had not time to do that. The peculiarity about this case is that the child hears about as normal children do at four years of age. It seems as though the ear is developing along the same line as in the average child of that age, though there are no external openings whatever. I would like to ask Dr. Dean if he attempted to explore the Eustachian tube in the ear that discharged?

**Dr. J. C. Beck**, Chicago, Ill.: I have some photographs here of cases like these that Dr. Dean has mentioned. Here is an interesting case such as Dr. Murphy speaks about. There is just a little rudimentary cartilage. The expression you can see is that of a mute, or a somewhat idiotic child. There is no reaction, try as much as you will. There is an absolutely dead labyrinth, and that, of course, precludes doing anything so far as the ears are concerned, except for the external deformity. An interesting thing in that case, if you will notice the fingers are tremendously long, all the digits, and the thumbs are almost as long as the fingers.

Another case of interest, as bringing out the question of facial paralysis in connection with this deformity, is a boy with complete facial paralysis, and another with partial. They are brothers. Both have occluded external meatus. These boys are absolutely deaf. That brings me to the point of inquiry as to what method Dr. Dean employed in bringing about the caloric action in the canal that was obstructed. I believe he said that about half the canal was blocked, and that the cut penetrated to the depth of the internal ear.

Another interesting case is this hanging ear, or lop-ear. This is a characteristic picture of these cases. As I have said before, my results have been of practically no value until recently. Whether they will hold out I do not know. I have found that by employing fascia from the trochanter region, and allowing it to become shrunken and hard, it will get almost like cartilage or bone. In this case I have made that operation. I have not yet a photograph to show the results, but it stands up almost as good as the other ear. I have it supported, of course, on props, but when the props are taken off I do not know how it will be. I have not even risked taking off the hard rubber form which I have in the ear to support it until it becomes hard. All these cases I have operated with good hearing. I have been able to follow Alexander's suggestions. If you will follow his technique thoroughly it gives you the best possible chance. He cuts over the aditus without disturbing the ossicles. I would like to ask the Doctor what method he used in examining the ears as to appearance, motility, etc., of the incus, tympanum, etc. I congratulate the doctor on his splendid report.

**Dr. W. B. Chamberlin**, Cleveland, Ohio: It was my good fortune about Christmas time to see this case with Dr. Dean. I do not think that any one talking with the boy would know that he had anything but normal hearing. This report is of special interest to me because of two such cases which I have under observation at the present time. One case is that of a girl, nine years of age, of perfectly normal development otherwise in every way. She has microtia of both auricles, with complete stenosis of both canals. In this case I had a very careful X-ray made, showing a practically normal mastoid process. In fact the mastoid was more than normally developed. I also had the patient examined by Dr. Todd, our anatomist, in order to get his opinion about the position of the facial nerve, as that is very important. We do not want to advise operation if we run the risk of an ensuing facial paralysis. Dr. Todd seemed to think that, in view of the normal development of the mastoid process, the facial nerve was probably normal in position.

The second patient is some thirty years old. There is a perfectly normal developed ear on one side, with microtia on the opposite side, with complete stenosis. In the normal ear there is some catarrhal deafness. One of the gentlemen remarked about inflation of the tubes. I catheterized both ears in this case. In the ear in which he had the microtia he had practically the same sensation as in the opposite ear. He could feel the air, as it seemed to him, just under the skin. That might be possible, as the patient is very intelligent. That gave me an idea to the potency of the Eustachian tube and the development of the middle ear. I hope to operate on both these cases later.

**Dr. J. A. Stucky**, Lexington, Ky.: I have a case similar to the one reported by the Doctor, a child, five or six years of age. On one side the ear is perfect, but there is no evidence of an ear on the opposite side. I am sure the child hears in the other ear, because I have used the alarm, and have spoken to her, and tested her in different ways. Now I am quite sure that since the birth of that child there has been some development of the cartilage, that is, it has grown some. The parents are also sure of that fact, but I have discouraged any attempt at surgical procedure in that case, as I still feel it would be a dangerous thing to undertake. I would like to ask Dr. Dean what he would think of such a case.

**Dr. Dean** (closing the discussion): In my haste to get through, Mr. Chairman, and not take too much time, I over-looked one thing, and that is this: The danger of exposing the glenoid fossa has been mentioned as an objection to operating on cases of atresia. In the second ear that was operated upon the glenoid fossa was exposed. There were no bad results from this.

In answer to Dr. Murphy's question will say that each Eustachian tube was badly strictured. Up to the present time no attempt has been made to dilate the tubes.

Dr. Beck called attention to the caloric test. One of my assistants as a matter of curiosity tried the caloric test before the operation with, of course, no result. The findings mentioned were secured after the operation.

In the operation the incus and the head of the hammer were well exposed. The incus on the left side was found to be apparently normal and movable. On the right side the incus was deformed. The ossicles were left in position. The aditus was closed by a fibrous membrane which appears as a concave drum head, if we might use that expression. Dr. Chamberlain, I am sure will bear me out, that there is a canal as large as the finger running down from the external ear to the bottom of the antrum. This is well covered with epidermis and there is no chance of it ever closing.

**Dr. Chamberlin**: Dr. Dean, don't you believe you would have gotten about as good results in that case if the ossicles had been completely removed, as you do in a radical mastoid operation? It seems to me that if you expose the ossicles to the extent that they were exposed in this case, you are going to disturb the relation to the ligaments, etc., and will get no function from the hammer, malleus and incus, no matter how able they may be.

**Dr. Dean**: Frequently in doing a mastoidectomy I have exposed the ossicles just as thoroughly as in this operation with an apparent perfect result with normal hearing following the operation.

In my judgment if you remove the ossicles you will have, because of the tympanic wall being covered with epidermis, an interference with the hearing just the same as you get in the radical mastoid operation. Here you have a membrane over the aditus which acts as a drum head, and the contents of the tympanic cavity are not disturbed.

# REPORT OF A CASE OF PURULENT LEPTOMENINGITIS OF OTITIC ORIGIN.

By DANIEL W. LAYMAN, M. D., Indianapolis, Ind.

I was called hurriedly on the afternoon of November 27, 1916, to see a patient in a comatose state. I was called because she was not responding to medical treatment and her physician thought the ear might have something to do with her condition. On my arrival, the only history obtainable was that Mrs. M., age twenty-three, had been in a semi-comatose state for the last three days. Her illness commenced about two weeks before with cold, followed by headache over the right side of her head. The headache became most pronounced, so intense that there was noticeable some mental disturbance. There was also a history of some nausea and vomiting.

Examination showed a well nourished woman lying in bed comatose. She could be roused only with difficulty, and showed distinct evidence of acute, marked meningitis—temperature 103°, pulse 125.

Examination of the ears showed slight moisture over both drum membranes. When wiped with a cotton probe distinct foul odor was noticeable, more marked in the right ear. On account of the meningitis and the serious condition of the patient I asked for a consultation with a neurologist to substantiate the diagnosis and consider with him whether or not an operation would be advisable. Dr. Neu was called, and his report is as follows:

"Complexion sandy, rather fleshy, lying in bed comatose. Patient can be roused and understands simple requests, but immediately relapses into unconsciousness, performing acts somewhat automatically. Skin is dry and rough; mouth dry; temperature, 103°, pulse, 120; respirations, 20. Shows marked cervical rigidity, pupils reacting to light. Kernig's sign on both sides, more marked on the right. Babinsky on left only; choking of both optic discs. An unsuccessful attempt was made to withdraw some cerebrospinal fluid. Later, while on the operating table, lumbar puncture gave a fluid with greatly increased pressure, turbid, slightly greenish yellow color, with considerable cellular deposit on standing. Microscopically, the deposit consisted of mononuclear and polynuclear cells more or less degenerated and diplococci, probably pneumococci."

The usual radical operation on right mastoid with an exposure and drainage of the dura in the middle fossa was determined upon, taking into consideration also surgical drainage of meninges on left side. The mastoid throughout was found sclerosed, and the cells contained some pus. The exenteration was done slowly because of the hardness of the bone and the close approximation of the lateral sinus to the antrum. The roof of the antrum was found necrosed and broken down, and on enlarging the opening an epidural abcess was encountered and cerebrospinal fluid in which there was pulsation. A segment of the squamous portion of the temporal bone was removed over an area about one inch and a half by one inch to expose the dura over the temporosphenoidal lobe. There was evidence of intradural pressure and an inflamed dura, especially over that portion of the dura contiguous to the antrum tegmen. A semicircular incision was made through the dura, and drainage instituted by gutta percha drains inserted fan-like well up between the dura and skull, especially in the subtemporal region; i. e., beneath the dura along the petrous portion of the temporal bone. At this stage of the operation Dr. Neu made a lumbar puncture and withdrew a creamy turbid fluid, the report of which has been given above. I did not do a radical on account of the serious condition of the patient. The hurry up sign was hoisted and I felt that something might go wrong in attempting fast work in a sclerosed mastoid and a lateral sinus jammed up against the antrum, thus jeopardizing the patient's chances of recovery, as Dr. Crockett states that "every minute over fifteen minutes for this operative procedure counts materially against the patient's chance of recovery," and I had already exceeded the time limit.

Convalescence was slow; both the constitutional and local symptoms subsided gradually, with the exception of headache which was rather severe for several days, being especially pronounced on the sixth day after the operation, when also a rise of two degrees of temperature occurred. It was necessary to change the outer dressings several times for two days as they were soaked with cerebrospinal fluid. The first complete dressing was made on the fourth day, but the subtemporal drainage was not removed until the second dressing on the fifth day. The patient was up and out of bed three weeks after the operation.

Dr. Neu's report of examination of patient two days after operation is as follows: "Spinal fluid with pressure still increased, but less so than previously—clearer, transparent, slightly fibril-

lated clot after standing several hours, the number of cells greatly reduced. Reduced Fehling's solution, slight increased protein content. The cervical rigidity was less marked. Kernig's sign was scarcely noticeable. There was still pain in the head, mostly over the area of operation. Patient had regained consciousness; optic discs still showed choking, the right more than the left."

Subsequent to the operation the following history was elicited from the patient. Had recurrent discharge from ears since four years of age. Discharge from right ear had been bloody and of foul odor the last few years and up to within a few weeks of her illness.

Careful treatment at the time of each dressing of mastoid and cranial wounds stopped the discharge altogether before the wounds were completely healed.

Cases of purulent meningitis recoveries after operation have been reported by American otologists during the last decade. However, these cures were not due to any well established method of operative procedure.

Of special interest are the series of cases and method of treatment reported before this Society last year by Dr. Crockett, of Boston.

I tried to carry out the surgical technique as laid down by Dr. Crockett. As I had not read the report in the Transactions only recently received, some of the details were overlooked. However, the essentials of my technique were as he had outlined, i. e., one and one half inch opening of the dura made at the bottom of the bone opening: The subtemporal drain inserted along the top of the petrous portion of the temporal bone. Dr. Crockett recommends that the lumbar puncture be made previous to making the dura opening, as the amount of pressure in the brain can be gauged by the finger tip pressed against the dura, thus grading the amount of fluid withdrawn.

Whenever this important subject is discussed some otologists put themselves on record as not having seen a case of purulent meningitis recover after operation. I had not, until this case, and notwithstanding the reports regarding self limitation and spontaneous recovery in diffuse purulent meningitis, I give credit for the cure of this patient to the method proposed by Dr. Crockett.

## DISCUSSION.

**Dr. Layman**, Indianapolis, Ind.: In presenting this case I wish to explain my position. When I accepted Dr. Brown's invitation to present a paper I announced an entirely different subject. I intended to present

a preliminary report on the "Results of Tonsillectomy in Systemic Diseases," but later on found that I could not present this in a satisfactory way because of lack of time. I did not want to be a quitter, and so I told the doctor I would report this case. On the way over here I was reading the Transactions of the American Laryngological, Rhinological and Otological Society in which Dr. S. McCullagh of New York stated that cases of acute leptomeningitis, on account of the rarity of cure, should be reported, so I feel a little easier about my position.

I really present this case for my own enlightenment, because I know in the series of cases which Dr. Crockett, of Boston, reported in 1909, there was doubt as to the diagnosis. The only procedure that I failed to carry out was securing a culture of the germs found in the spinal fluid. That was not done. There have been various new methods proposed from time to time and I would like to hear from men of experience, Dr. Beck, Dr. Pierce, and others. I remember at the Section last year Dr. Dench said that a large decompression operation in a number of his cases had been successful. Dr. Crockett presented two cases which recovered without operation. Now the question is when are we to do the decompression without opening the dura, when are we not to operate at all, and when are we to open the dura and go the full limit?

**Dr. J. C. Beck,** Chicago, Ill.: We all know the discussions that go on year after year. Dr. Pierce always leads the discussions, believing that any one can get well from meningitis. I think he is right absolutely, except as to his statements in regard to operations.

Make up a series of 104 cases of clinically diagnosed septic perimeningitis that have recovered with nothing more than spinal puncture. You will remember when Professor Jansen visited this country he read a paper on that subject. However, Dr. Pierce is absolutely right that they generally recover. They are not a fulminant meningitis but septic cerebritis. These are the cases in which Dr. Day has shown specimens, in which there is fibrinous exudate at the base of the brain with septic masses running into the brain. That, of course, was not true of Dr. Layman's case. His case was a serous meningitis proper of the fulminant type, just at the moment when, as Dr. Kopetzky says, the relative acidity of the brain is increasing, and the protective substance of the brain is used up, that is the important time in these cases, and the doctor operated in time.

The decompression operation just alone is very important, and the cases do get well by decompression. It is by increased pressure that the injury is caused to the brain, and then you get secondary infection into the brain. Kopetzky has brought that out in his work. His work has been attacked, but he has proven his point over again, and I think the proposition is settled, so far as I have heard from New York at least. As I understand it, the subject is to be brought up again at our next annual meeting.

Now then, this was a case of diffused serous meningitis and even if bacteria were found it is not yet a case of septic meningitis, as I understand it, a condition in which the brain is already affected. Of course that is a very important point so far as recovery is concerned. When once the cortex is involved, if the patient does recover he is an imbecile. There were cases reported years ago of meningitis secondary to the ears. Dr. Macewen's work, of Glasgow, shows that this class of cases existed before his classical work on this subject appeared. The point is that the operation was done early. You should not overlook a thorough bacteriological examination. Give data of the fluoroscopic picture, of the moisture in the ear, the foul odor. Another thing, why was one side only operated upon? If one side sufficed it is certainly interesting that one side should be operated upon and the other get well, the other ear and also the brain. In this connection I would like to mention an important fact in regard to operating on one side of the head, and the other getting well. I recently operated on a subject

who had acute bi-lateral otitis media, with fulminating symptoms. The bi-lateral mastoid popped in 24 hours. I operated the discharging ear and found complete destruction of the mastoid, with the sinus exposed. The other ear in practically the same condition. Gas had to be given. The child had a hemorrhagic nephritis, and a bronchitis, and was supposed to have an undiagnosed erysipelas. Now then I operated the one ear under gas anesthesia but had to stop because the child was showing symptoms of heart dilatation, and the expert anesthetist said that he preferred not to give this anesthetic any longer. I had expected to operate the other ear. Following the operation on one side the other ear rapidly got well, the ear became dry, and hearing is practically normal. I was astounded at this rapid change. The septic process has been arrested. If the child gets well, but shows any tendency to a flareup, certainly that mastoid should be opened and cleaned out. I think there is a focus which may light up at any time.

I wish to return to the subject of meningitis—the question of treatment purely from my own experience. I have done a number of these experimental operations of Haynes. Had one case get well, a pneumococcus meningitis. It is on record. It was a serous meningitis in which the bacteria was obtained, not a septic process. I have operated after the method of Cushing. If Dr. Layman will look up the Transactions he will find I discussed a paper saying I had operated on a case of that kind. I know nothing about Dr. Crockett's method of operating.

I think so far as operating on meningitis or abscesses, the thing that we can look forward to as giving better results in these grave conditions are the two stage operations. If we have abscesses, or acute processes, like possibly an acute sphenoidal abscess, make a large decompression, wall off first between the bone and the dura, then incise and pack around, as the surgeon does in the appendiceal region, put a loose fluff of gauze in, and then allow nature to work the pus off that decompressed area. The patient will be better off than if you look for the abscess. Now instead of draining the cisterna magna, which is some operation, it is easier, I think, if the spinal puncture drainage is not sufficient, to make the operation recommended by Kanavel, namely, the corpus collosum drain. That is a very simple operation. You make a small opening in this area, a small piece of bone is taken out, a specially devised needle, blunt pointed, is passed as far as the longitudinal sinus, then turned on its axis so that the point is pointing downward, getting down to the fornix, between the meninges, between the two lobes of the brain, then on top of the corpus collosum thereby obtaining drainage. A quantity of fluid is allowed to escape, and then you repeat it. The spinal puncture procedure is to reduce pressure, so that there will be less chance of a cerebral hernia occurring.

**Dr. W. E. Sauer**, St. Louis, Mo.: I would like to mention a case of meningitis following a case of otitis media, which I reported three years ago. In this case we did a number of lumbar punctures and were able to grow streptococci from the cerebrospinal fluid. The patient ran a very slow pulse and had a very marked increased intracerebral pressure. The consulting neurologist suspected a brain abscess and we punctured the temporosphenoidal lobe, making four punctures, but no abscess was found. We made three punctures on the cerebellar, with negative findings. The patient finally recovered and left the hospital in apparently a normal condition. I did not hear from her until three weeks ago, when I received a report that she had died from a ruptured temporosphenoidal abscess.

**Dr. J. A. Stucky**, Lexington, Ky.: I want to congratulate Dr. Layman on his prompt operation, and the result obtained. I am glad Dr. Beck thinks there is such a thing as serous meningitis. At the meeting of this section in Indianapolis, a few years ago, he did not admit it. The first stage of meningitis is the serous stage, I am convinced. I quite agree with Dr. Beck that after it becomes generally septic we have septic cerebritis and the patients do not get well. If we could get at

these cases earlier, and do as Dr. Layman has done, I believe we would save very many cases of meningitis.

**Dr. Beck:** I want to make a correction. I do not believe there is any localized serous meningitis.

**Dr. Layman:** Dr. Beck asked me why one side was operated upon; in other words, why I made a choice of sides. There was a history of more pain on the right side, on the side operated upon. The husband stated that she had had more trouble with that ear in the last few days. That brings up the point that in case both sides had been found equally disturbed, had there not been pain or any definite symptoms pointing to the one side, would it have been justifiable to have operated on both sides at the same time in as serious a case as that, without having anything to go by?

**Dr. Beck:** The proof of your case is the pudding.

**Dr. Layman:** We had considered that, and the neurologist had advised it, and I presume it would have been the right procedure had we not localized the offending side at the time of the operation. I failed to give a definite description of the appearance of the dura membrane. There was moisture covering the ear drum, with a foul odor, a marginal perforation, and some congestion above with bulging. That was incised thoroughly at the time of the operation.

## THE SERUM TREATMENT OF FETID CORYZA AND OZENA.

By J. A. STUCKY, M. D., Lexington, Ky.

Mr. Chairman and Gentlemen: I simply desire to continue my report made at the Annual Meeting at White Sulphur Springs. I have been using the serum treatment as advocated by Dr. Horn, and since then have had five cases. All of them were typical cases of ozena and fetid coryza, the disease which has proven a black eye and a reproach to rhinology, in that we have never been able to do anything for it, other than give temporary relief. All of the patients were female—three young ladies whose ages varied from 18 to 23. Two were middle-aged women, the older 51, the younger 44. I have been able to keep these cases under observation now for eight months, and I have been more pleased with the results from this treatment than anything I have ever used. In a letter received from Dr. Horn the other day he tells me that they have named this serum bacillus the rhina ciptiens. This bacillus is the bacterial and etiological factor in this disease.

I began the treatment, using it a little differently from the method used by Dr. Horn. I began with two hundred million, and repeated that dose in three or four days. I have allowed no treatment after the first treatment that I give, that is, no local treatment. I remove all the crusts, being careful to remove any interference with drainage. If there is an interfering turbinate or a spur I get rid of it. In every one of these cases you could detect the odor within three or four feet, a most horribly foul odor. One of the patients, whose case I reported at White Sulphur Springs, had traveled not only all over the United States, but all through Europe seeking relief. She was one of the belles of the Blue Grass, but yet she could not go into society because of the disagreeable odor. This was of course humiliating. After the fourth treatment there was no odor. It has now been six or seven months since I began the treatment, and so far as I can tell she is entirely free. She insists on having a treatment once a month. I began by giving two hundred millions, repeated in four days, and then added two hundred millions as laid down by Dr. Horn, until giving two billion. Two of the patients had a slight chill, with headache, and backache, but that is all the reaction I have met with. I thought that was probably a coincidence. My son is now using it in his

clinical work, and reports gratifying results. I believe we are at last on the right road, with a remedy that will enable us to control this horrible disease. At any rate, the indications are favorable. I cannot say too much about it. I am now able to get all the vaccine I want. Before I had been unable to get it for more than seven cases, but they tell me now they can furnish all I want. The first results seen, the crusts begin to get small, the patient is able to cleanse the nose by blowing, and does not have to resort to a douche or atomizer, or anything else. The first evidence of improvement is that the odor goes, and then the crusts disappear.

## Report of Case of Total Deafness Resulting from a Myringotomy.

Six weeks ago a young woman, school teacher, was referred to me by a physician in an adjoining county. She came in with the earache. Was totally deaf in her left ear. She reported that two weeks before that she had suffered from a very severe earache, and the drum was red and bulging. An anesthetic was given in order to make a myringotomy. The patient woke up totally deaf. She had vertigo and nausea for several days, and was confined to her bed. The earache was relieved, but she is totally deaf with no labyrinth or vestibular reaction.

Just what occurred to cause the deafness we can easily surmise. The physician who did the myringotomy makes a specialty of eye, ear, nose and throat diseases, having taken a course of six weeks in these branches in a New York school from which he has a diploma. Of course, this case may be an accidental one with an abnormal anatomical arrangement of the interior of the middle ear cavity. This is the third case I have met with in the last nine years and is another unmistakable evidence that any operation within the middle ear is not a simple one. There is room for thought not only for the teacher of Otology but for the young Otologist as well.

### DISCUSSION.

**Dr. Thomas J. Harris**, New York: This report of Dr. Stucky's (speaking entirely of the paper, and not attempting to refer to the case which he has mentioned), is decidedly interesting to me, interesting in many respects, but the main point is the favorable results he is reporting from the use of vaccines. I only wish I could follow him. That subject is as interesting to me as it is to him, as it is to us all. If there is anything in vaccine for the treatment of ozena, we want to know it, all of us. We have been trying in New York for the last year, more conscientiously than ever before, to carry out the work of

Horn. We have been taking every case in the service and making a careful bacteriological examination of the secretions, and making complete X-ray examinations of the sinuses, and we have been getting results quite at variance, I am sorry to say, with what Dr. Stucky has secured. In the first place we have not been able to identify the Perez bacillus in anything like the proportion of cases you might expect. In not more than 25% of our cases have we been able to discover the Perez bacillus, in other cases the Friedlander. Our bacteriologists are in the same position as that reported in the Archives of Laryngology last year, by one of the workers in Copenhagen, namely, that they doubt very much whether there is organism such as Perez has discovered. In fact, his points of identification are so vague, that it is very difficult for our bacteriologist to run them down. Our results do not bear out Perez's or Horn's statements. As pointed out by Dr. Stucky, Dr. Horn has begun to revise his own views, and in his last pamphlet which was sent out four or five months ago, he made the statement that he no longer believed that the odor could be regarded as characteristic. He also made the statement that he was satisfied that the vaccines would not cure, but only relieve, and that it would be necessary always to continue the use of the vaccines. In regard to other findings in our cases: We have found, with one or two exceptions, there was disease of one or more of the accessory sinuses. In only one case under my care where we have given vaccines, have we had such results as Dr. Stucky reports. It has been must discouraging in carrying out the vaccine treatment week after week (considering the work that Dr. Stucky has been doing, and hearing what Dr. Horn has said about his successful results) to find that the crusts do not disappear. Another interesting point in regard to Dr. Coffin's work. It has been found by opening the antrum in three or four cases recently, and draining, the odor has disappeared. After working that out himself, Dr. Coffin found that Dr. Horn speaks of that. He says he has made that observation in his work.

I am really interested in knowing how the workers in the different parts of the country seem to succeed so differently. It cannot be the explanation which I heard the other day, in regard to the famous visit of Dr. Hooper of Boston. He came to New York many years ago with a number of large bottles containing adenoids. He pointed to these and said that that was the result of operations on Boston children. One distinguished New York laryngologist got up, scratched his head, and said, all he could say was he didn't think New York and Boston children were alike. He didn't think you could get anything like that out of New York children. Whether Dr. Stucky and Dr. Horn's noses are different from ours, I do not know, but I wish they would come on to New York, and show us how they secure their results from the vaccines.

**Dr. E. T. Senseney**, St. Louis, Mo.: During the last year I have had three cases under observation. In only one, a child, have I seen any benefit at all. This child, after repeated injections extending over a period of six months, improved. The treatment was allowed to lapse for several weeks, and the trouble returned. However, as long as the treatment is continued the crusts are better. In the other two cases there was no benefit whatever. I think, if I am not mistaken, that has been the general experience in St. Louis.

**Dr. H. B. Harris, Dayton:** I want to emphasize what Dr. Harris has said as to disease of the sinuses. Five years ago I began to do work along this line from an operative point of view. I took the stand that there is always some persistent sinus disease. In every one of my cases I found that there was some decided disease in some or all of the sinuses: first and most frequent the ethmoid, then the ethmoids and sphenoids, next the ethmoid, sphenoid and frontal, and lastly the

antrum. I have operated a good many cases in which I have used the vaccines subsequently, and have noticed that not only do I get no bad results from the operation but the general conditions have improved. I do not believe it is possible to do any draining in the ethmoids without scooping out entirely and absolutely. Within the last year I have used some of Dr. Horn's vaccines after operations, and have gotten the results which Dr. Stucky mentions, but I get no benefit before operation. I believe that the use of the vaccine should be preceded by a clearing out of diseased sinuses if they are present. You get better results from the vaccines after operation.

**Dr. Harold Wilson,** Detroit: We have had four cases under our observation for more than a year. Some of them have been treated with a mixed bacillus vaccine, and others with an "Ozena (Perez)" vaccine. We have been entirely unable to see any material change in the symptoms.

**Dr. T. J. Harris,** New York: Just a word, Mr. Chairman. Dr. Dwyer, who does the bacteriological work at the Manhattan, showed me a letter which he had received, which reads as follows: "Dear Doctor: I want to tell you how much better my child is. The odor has entirely disappeared, there are no longer any crusts in the nose, and we are eternally grateful to you." Dr. Dwyer has been giving the girl an autogenous vaccine for a number of months; showing an ordinary streptococcus infection. He simply made up a vaccine and got this result, quite bearing out the idea that it is not a special organism which causes the trouble.

**W. B. Chamberlin,** Cleveland: I would like to add a word along the line mentioned by Dr. Wilson. For a number of months I have been treating some of these cases, mostly in children, and have used the vaccines made by the Parke, Davis people. In addition to the vaccines I have used a 5% solution of scarlet red in an emulsion of quince seeds. It is therefore hard to tell what gives the results. I have had good results, so far as the disappearance of the odor is concerned, together with a marked diminution of crusts. The only report I could give would be that there was marked improvement—no absolute cures.

The point which Dr. Stucky failed to mention was in regard to the regeneration of the epithelium. I have seen no attempt at regeneration at all. In other words, when the scar tissue once forms it remains. There is no attempt at restoration of the initial mucosa. The injections were given twice a week. There was one rather interesting observation I made. We have a large Italian colony in Cleveland. There is a corner of town known as Little Italy. I had three Italian children under treatment. One was beyond the school limit, and was working in a factory; the others were in school and I had to see them at times that would not interfere with their school work. It made me wonder if this disease was more common among Italians than other nationalities. The two cases were in the same room at school, and each child accused the other of giving the disease to her.

**Dr. Harold Wilson,** Detroit: As a mere suggestion, I would say that any bacterial protein will do the same thing as Dr. Horn's vaccine or Parke-Davis's specific vaccine. I am rather of the opinion that it is not the specific reaction which takes place when the local conditions improve, but it is a protein reaction. The gonococcus, the typhoid bacillus, or most anything you have on hand will answer as well.

**Dr. J. C. Beck,** Chicago, Ill.: I think this discussion as to a bacteriological antidote for this disease is an absolute waste of time. I do not think the bacteria have anything to do with this specific organism. I think the bacteria are present because it is a bone disease, there is the degeneration of the membranes. There is a putrefactive

organism present, just as we find it present in the teeth, the tonsils, or any cavity in which you have decomposition going on. If you take a section of the atrophic inferior turbinate—and it probably takes a little courage to say one has removed an atrophic inferior turbinate to see what it looks like, and if Dr. Pierce is listening he will say it is not right to do that—but I have removed a particle of the inferior turbinate to see what it is made up of. You will find that the primary change, early, in atrophic rhinitis, is a rarified process, an osteo-porosis of the bone with atrophy. The mucous membrane glands are present, there is an outpouring of very sticky tenacious material, you will find bridges of this material between the septum and the turbinate; later on there is shrinking of both the mucous membrane and the bone, and there is an osteo-sclerosis until finally the whole process has been destroyed, and the mucous membrane with it. It has been shown, years ago, that it is a metaplasia of the epithelium, and that a systemic condition is the underlying cause. He said that girls have rhinitis mostly. My belief is that this is a systemic condition. I believe it is just the same in this bone change as in the calcareous bony change which we find in the internal ear—an osteo-porous spongification with finally hardening.

**Dr. Stucky:** What is the difference between the pathology of atrophic rhinitis and ozena?

**Dr. Beck:** Ozena is only the name of the smell. It has nothing to do with the pathology. Ozena is not the disease, it simply refers to the smell. Atrophic rhinitis may be genuine ozena, or an atrophy secondary to sinus disease. Atrophic rhinitis is secondary to infection, to a dribbling of pus over the inferior turbinate.

MEETING OF THE WESTERN SECTION, HELD IN COLORADO SPRINGS, COLORADO, MARCH 17, 1917, UNDER THE CHAIRMANSHIP OF DR. JAMES A. PATTERSON, OF COLORADO SPRINGS.

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## SOME INTERESTING LABYRINTH AND EIGHTH NERVE CASES.

By H. B. GRAHAM, M. D., San Francisco, Cal.

We have all of us in our examinations of the eighth nerve neuritis cases been struck with the multiplicity in the causes and in the variation in the symptoms. The diseases of all the cranial nerves seem to follow about the same course exhibiting symptoms and an ability to recovery varying according to the function of the nerve, the quality of the cause of the lesion and the intensity of the attack. I have in a former publication pointed out these qualities in respect to the sixth nerve which have been so ably reviewed by Passow and others and they have been referred to in the description of the peculiar menieriform polyneuritis of the cerebral nerves described by Fränkel Hochwart. For the sixth nerve the causes have been proven by autopsy to be first pressure, as from a tumor; second, serous meningitis from middle ear infection; third, the effect of suppurative processes at the apex of the petrous portion of the temporal bone; fourth, toxic, as from lues, lead, arsenic, alcohol and tobacco or from intestinal toxemia. We can readily see how these same causes might act on any cranial nerve and especially on the eighth but in addition to these we have arteriosclerosis, heart lesions and a nerve exhaustion appearing as prominent factors with the eighth, factors which have not been mentioned in other cranial nerve neuritides. The eighth being so peculiarly mixed in its function and so intimately connected with other portions of the nervous system, exhibits more pronounced susceptibility to remote influences and shows a larger variety of easily demonstrable symptoms than do other cranial nerves and becomes a very important though much neglected factor in the diagnosis of various diseases. This group of cases exhibits a partial or total, temporary or permanent deafness which has a tendency to reeurrence during which the functional testing may give anything from a middle ear disturbance to that of the typical

lesion of the internal ear. Accompanying this may be an apoplectiform disturbance of the vestibular apparatus as exhibited by dizziness and disturbances of equilibrium, inequalities in the after turning nystagmus, caloric and other galvanic reactions and interference with the kinesthetic sense. Either the cochlear or the vestibular symptoms may be uppermost or one symptom or the other alone affected making the lesion as a whole overlooked or referred to other parts of the body as is often the case when the patient is superficially examined.

To illustrate:

*Case No. 1.* A laborer, 30 years of age, appeared at the Stanford Clinic complaining of an inability to navigate properly and with a ringing in one ear. He appeared to be drunk and was examined with that in view with a result which entirely exonerated the alcohol. His right ear was non-reactive to caloric and he was deaf in this ear. There was a spontaneous horizontal nystagmus to the opposite side. These symptoms continued with diminishing intensity for a month while the patient was in bed and when he left the hospital were still present to a marked degree. Three months later he returned and announced that a week following his dismissal from the hospital he had found that his liver was out of order and had taken some China medicine with prompt secession of all symptoms. While he was in the hospital exhaustive examinations were made of the urine, feces, blood, spinal fluid and complete physical examination by several internists and at no time could any assignable cause be give for the neuritis nor was there anything in his history which would point the way.

*Case No. 2.* F. E., a laborer of 35, came to me complaining of a dizziness so great that he had been in bed for three days. He gave a history of having had three years previously a headache lasting two years followed by a paralysis of the sixth nerve. One month before the paralysis came on the headaches became less to cease on recovery from the paralysis one week later. On examination I found perfect hearing in both ears, no tinnitus, functional tests normal save a slightly shortened bone conduction, knee jerks were O. K., but the patient says they were gone one year before and there is a six year old child in whom I found an absence of knee jerks but who was otherwise normal as was also his wife. The patient exhibited a constant spontaneous horizontal nystagmus to the right, an absent caloric on the left; turning to the right produced a few jerks, turning to the left gave an extra nystagmus of fifteen seconds to the right. The next day the spontaneous nystagmus was much less and turning in either direction gave a marked nystagmus to the right, turning to the right a nystagmus of fifteen seconds and to the left a nystagmus of twenty-five seconds. There was a very slight caloric from the right, none from the left ear; hearing was perfect so that the right vestibular nerve was now involved with the left. Five days later the spontaneous nystagmus was gone, caloric was slow from either ear, turning always gave a nystagmus to the right of the same value as before. One and two months later the caloric was prompt from the right ear and absent from the left, the turning nystagmus was normal in direction, of small excursion and of a value of twelve to fifteen seconds. There was a negative blood, Wassermann, and

the urine showed an excessive amount of indican when he first came in. I placed the man on mixed treatment but do not consider that it was at all established that there was any luetic influence in the case and the man himself gave a very clear negative history.

*Case No. 3.* An unmarried clerk of 30, a young woman under an excessive nervous strain without a history or signs of lues, who, periodically with six or eight months intervals, became dizzy for about a week, had to go to bed and exhibited a nystagmus to the left with an exaggeration on movements of the head, absent caloric on the right and perfect hearing.

*Case No. 4.* An active business man of sixty with an arteriosclerosis negative Wassermann, a history of having previously had a slight cerebral hemorrhage and frontal headaches for twenty-five years, came complaining of unsteadiness in his walking. Functional tests gave Weber unlateralized, Rinné positive and Schwabach slightly shortened, watch not heard, forks well heard, monochord at twenty-three. On turning to the right there was little or no nystagmus but marked dizziness. On turning to left there was a nystagmus to the right of twenty-two seconds but the patient was not so dizzy. There was a slow caloric with small symptoms from the right. In other words, there was an incomplete neuritis of the left vestibular nerve due to an arteriosclerosis and on testing this left organ by turning there was a minimum nystagmus but marked dizziness and by the caloric test both a short nystagmus and minimal subjective signs.

I have selected these cases from varied classes and ages, probably none of them are luetic, no two exhibited similar nystagmus; none of them happen to have symptoms from the side of the cochlea but these are well known and frequently found so I need not burden you with them. What role interference in intestinal gland secretions have in the production of these disturbances is not known but there seems to be evidence pointing to them as the immediate cause in many cases and the subject is under study at the present time.

I wish now to call your attention to a case exhibiting a peculiar fistula symptom for which I have no explanation.

*Case No. 5.* A telegrapher of 35 gave a history of having, seven years before, had a dizziness in blowing his nose which lasted for four or five minutes and no further disturbance until about three days ago when he noticed the same thing. He had a marked coryza when seen; the day before the telegraph instrument caused a sensation of pain in the right ear. There was a hissing noise in the right ear, both ear drums were slightly injected but transparent. The right drum showed some clouding at the inferior half and an atrophic area in the superior posterior quadrant. The left ear was normal, the right showed slight shrinking for the watch and whisper, seventeen and a half for monochord, fourteen and a half on the left. Weber was to the right; there was no fistula symptom. On politzerizing this patient, a marked rotary nystagmus occurred in both eyes, at first lasting a couple of minutes to the left and then changing to the right and lasting another two or three minutes, the patient broke out in a profuse perspiration and became markedly nauseated, this lasting for an hour. The whole phenomenon was repeated

three days later. Ten months later the patient heard normally in both ears, monochord fourteen. There was no disturbance on politzerizing. I present this case without comment for your discussion.

*Case No. 6.* There is another type of case which I wish to call briefly to your attention and that is an acute temporary disturbance of the cochlear apparatus due to nerve exhaustion or to an acute infection in which there is an acute shrinking of the high tones and a shortened bone conduction both of which pass off in due time, after a rest or recovery from the infection. I first noticed this in an overworked insurance agent who had had some disturbance in hearing before I saw him. He came to me complaining of an impairment of hearing and in the course of a week I watched and demonstrated to others the rapid loss of perception for the monochord until he could no longer hear it at all with the rub and only partially with the bow. This cleared up entirely after a couple of weeks' rest from a very nerve racking occupation.

*Case No. 7.* The next case of this type I saw was an infection with so-called influenza (not a bacterial diagnosis) where there was a shrinking of the perception for the monochord to thirty or less and there was a marked shortening of bone conduction, both of which cleared up in a week.

Rapid involvement of the labyrinth in an acute suppurative process is not frequent but does occur as illustrated by the following case.

*Case No. 8.* A 34 year old office employee while in perfect health contracted a fever and became dizzy. A physician said on the following day that he had tonsillitis, although the patient was not aware that there was any disturbance in his throat. On the third day the left ear drum ruptured spontaneously and the ear continued to suppurate until seen by me five weeks later. For the first three weeks there was an extreme dizziness and nausea with vomiting so much so that he thought he was going to fall out of bed. The vomiting ceased after the first week. My examination showed an acute mastoiditis with a non reactive labyrinth throughout and a Neuman labyrinth operation was done with no dizziness immediately succeeding the operation. The organism was a capsulated streptococcus. The invasion must have taken place at the very beginning of the infection but by what route I can not say. He evidently had not previously had any disturbances of the ear and no fistula could be demonstrated in the labyrinth capsule at operation. It is problematical as to whether drainage of the antrum on the second day would have saved this labyrinth but I am inclined to believe that were I to see a case exhibiting these symptoms again I would strongly advise such an early procedure.

*Case No. 9.* Lastly I wish to relate a case exhibiting the aural calamities that an individual may be subject to. A man having fallen seven floors, three years and a half ago, sustained a right suppurative ear. Two months later he was sandbagged and was dizzy for four days, so much so that he could not work. This passed off partially, the patient becoming steady by day but not at night. After the sandbagging he was stone deaf in the right (suppurative) ear as determined by a doctor. At the examination it was determined that there was considerable impairment of hearing in the other ear, the high tones being short. The caloric in this good ear was absent, there was no nystagmus on turning in either direction. The patient pointed promptly without error at the object after turning. I take it that the man had a suppurative laby-

rinthitis in one ear and a concussion affecting the vestibular apparatus in the other. A labyrinth operation on the right was succeeded by no dizziness. The patient exhibited the interesting phenomenon of being unable to orientate himself in space as when diving in a tank of water.

In discussion, Dr. F. R. Spencer suggested that there was a congenital dehiscence in the promontory, in Case No. 5, the case with a fistula symptom ou Politzerizing.

Doctor E. R. Lewis called attention to the work of Dr. Jones of Philadelphia who has demonstrated that the motor and sensory portions of the vestibular nerve divide to reach the cerebrum and cerebellum at different levels in the central system, thus giving a chance for a marked difference in the symptoms as well illustrated by case No. 4 where there were marked sensory signs on turning and small motor (nystagmus) signs.

# TRANSILLUMINATION OF THE LARYNX AND UPPER TRACHEA.

(First Communication.)

By FRANK R. SPENCER, M. D., Boulder, Colo.

In June 1916, while the guest of Dr. Joseph C. Beck, at the North Chicago Hospital, I was invited to look into the larynx and trachea of a patient suspended with Lynch's suspension laryngoscope. The Beck light, on a stand, was at the patient's left shoulder and the shade so placed that much of the light was thrown on the left side of the larynx and upper trachea. I chanced to view the interior of the larynx and trachea the instant before my head mirror could throw the light into the upper respiratory tract, and I had my attention immediately attracted by the readiness with which the interior was illuminated by the light on the stand. I called Dr. Beck's attention to this and the word "transillumination" quickly suggested itself to his active mind. The thought that I had discovered something new occurred to me, but upon looking up the literature I found I was mistaken.

Whether *this particular method* of transillumination is of any real value and whether this method of using it is original I cannot say, but I think it is. However, transillumination of the larynx was first practiced by Czermak,[4] but was later perfected by Voltolini[6]. Freudenthal states, in a letter, that he has had experience with transillumination of the larynx and that he was the first one in the United States to demonstrate the method. He has written articles upon this in German[2] and in English.[1,3]

I shall present this method for what it is worth and let the future decide its real merit and permanent place in laryngology. However, I am afraid transillumination of the larynx and trachea has only very limited practical value when compared with transillumination of the maxillary sinuses, because it is so easy to obtain a good view of the interior of the former and so difficult to do so with the latter.

The following are the methods of applying transillumination to the larynx:

1. Voltolini[6] used a metal covered electric light for condensing the rays of light on the exterior of the larynx.

2. Freudenthal[2] improved upon Voltolini's model of laryngeal transilluminator and applied it either at the pomum Adami, or near the cricoid cartilage, and introduced the heated laryngeal mirror into the dark pharynx so as to obtain a good view.

3. Cheatham has used a De Zeng transilluminator, but he doesn't say just how he applied this. Several other laryngologists have used this test, or method of examination, in different ways, judging from their replies to my circular letter of inquiry to all the Fellows of this Society.

4. The Beck light, on a stand, may be used to good advantage to light the larynx, while the patient is suspended, as I mentioned in the first part of this paper. Whatever the method of applying this, the principle is the same.

Transillumination, even in normal patients, should be of value for inspection of the larynx and upper trachea. In cases of benign growths, it should give a fairly definite idea of the amount of inflammatory thickening about the point of attachment. This will be of even greater value in cases of malignancy or suspected malignancy. The same may be said of tuberculous or syphilitic ulcers, lupus, chondritis, perichondritis, etc. Freudenthal, in a personal communication, relates the following: "I recollect one case which I saw, together with Dr. Lefferts and the late Dr. Asch. It was a man with terrific stridor. Both these colleagues had made the diagnosis of aneurism, while my diagnosis was carcinoma of the trachea. A dark shadow was plainly seen on transillumination. At the autopsy both conditions were found, i. e., a cancer, on top of which (or on the side, I do not recollect particulars) was an aneurism." As this was prior to the bronchoscopic and suspension laryngoscopic era in medicine, such a test was of more value then than now, other things being equal.

As the test can be made during either suspension or direct laryngoscopy, or by a modification of indirect laryngoscopy, it can't be used as easily as transillumination of the maxillary sinuses, for instance. Direct or indirect inspection of the mucosa of the upper respiratory tract, by any of the standard methods, is of greater value than transillumination, especially since indirect inspection is comparatively easy. On the other hand, we should use in laryngology, as in the other specialties, all the tests at our disposal, if we cultivate accuracy in diagnosis. The average case may not and probably won't require it, but the difficult ones will, especially if direct or even indirect laryngoscopy is very difficult

or for any reason impossible. I have in mind two patients with ankylosis of the mandible, as a part of a general joint infection due to tonsillitis, in whom it would be difficult or even impossible to examine the larynx except by transillumination. In fact, the ankylosis is so pronounced that it is difficult for them to masticate properly. While such cases are the exception to the rule, they do occur.

Schmidt,[7] however, has the following to say: "For laryngeal examinations, in which the transilluminator with a cover, as in frontal sinus examinations, is placed anteriorly on the larynx, I regard the method as without value. Besides, it is very difficult to place the mirror in the correct position in the dark pharynx. It is true that a transilluminated larynx gives a beautiful picture, but I have not obtained any practical results from the method."

Roth[11] doesn't favor transillumination of the larynx, as he believes the test isn't practical.

Dr. H. Arrowsmith writes as follows in a letter: "Transillumination of the larynx was done, to a very limited extent, twenty-odd years ago. I tried it out, casually at that time, but with others discarded the procedure as having no practical value."

The normal larynx shows shadows cast by the thicker portions, such as the true and false cords and the cricoid cartilage. The thyroid cartilage offers, of course, the greatest obstruction to the rays of light. The rings of the trachea cast very definite shadows.

The ventricle of the larynx, the crico-thyroid membrane, and the membrane extending between each two adjacent tracheal rings admit more light than the thicker structures, hence, at any of these points, inflammatory thickenings, benign or malignant growths will show most distinctly on account of the shadows which they cast. The same may be said of the membrane extending from the lower border of the cricoid to the first ring of the trachea.

During the past fifteen months, I have had under observation a papilloma of the larynx in a man, age 41. This growth has returned twice following removal by suspension laryngoscopy. The papilloma originally sprang from the left ventricle, but later seemed to be attached to the inferior surface of the false cord too. This casts a distinct shadow by transillumination, during suspension laryngoscopy, and shows well the inflammatory thickening at the point of attachment. While the pathologist reported a suspicion of malignancy from the second papilloma removed, I don't believe it is malignant, but the shadow cast is due purely to the thickened tissue.

Cases examined with the bronchoscope or esophagoscope for a foreign body or a stricture, it is needless to say, show only the normal shadows. Imperatori states, in a letter: "I have noted the light passing through the cricothyroid membrane while suspending the patient and with the long lighting attachment within the larynx." Since I have not had an opportunity to use this in a single case of malignancy, I can't speak from experience of its value here.

J. Solis-Cohen, in a personal communication, writes as follows: "I have tried transillumination of the larynx and trachea, but got very little satisfaction from it. The parts looked like translucent candy and it was not possible to outline growths and structures, so I gave it up. I do not think I ever wrote upon the subject."

Schech[5] makes the following interesting statement: "The transillumination of the larynx, first practiced by Czermak, and later being perfected by Voltolini, has no diagnostic value."

Dr. Frank E. Miller writes as follows: "I have had at least fifteen years experience with transillumination of the larynx and trachea. In each I find it especially helpful, where there are gouty deposits and tuberculous combinations. It has proved extremely satisfactory and has been particularly helpful in diagnosing cancer cases, by exhaustive processes. At the present time, I have three cases of gouty deposits of the thyroid and arytenoid, where the above had not been employed, causing improper recognition and tracheotomy. In a number of cases where this has shown simply a shadow overlying the thyroid gland, the penumbra has resulted in serious errors of diagnosis."

Frankel[8] has referred to transillumination of the larynx in Simon's handbook of laryngology, but mentions this very briefly.

Dr. Norval H. Pierce states: "I have had some experience with transillumination of the larynx and trachea, and now have a paper in preparation on the subject. This procedure has been used before, as a paper was read on the subject before the Belgian Laryngological Association."

A. L. Benedict[9] has suggested the use of transillumination of the upper respiratory tract.

William Cheatham, in reply to my letter of inquiry, states: "A few years ago I became interested in laryngeal transillumination and used De Zeng's frontal sinus transilluminator. I found the study very interesting."

Dr. Lee M. Hurd writes as follows. "Four years ago, while observing a bronchoscopic tube, being passed in a darkened room, I noticed the translucency of the light on the neck and chest wall as

the tube was passed, and later reversed the idea of light outside, using mirror to examine larynx and trachea. I have used the method for observing the contrast and to out-line growths in the larynx. Lennox Brown used a similar method 30 years ago."

To quote further from Freudenthal[1]: "Now, to get a view of the interior of the larynx we apply this lamp to the external surface of the neck, either at the pomum Adami, or near the cricoid cartilage, and introduce the heated laryngoscopic mirror into the dark pharynx, and thus we obtain a splendid view of it. But this view is quite different from that ordinarily seen. The first thing that strikes the observer is the disappearance of different colors. The whole larynx shows a reddish tint of varying intensity. When we place the lamp at the level of the incisura thyreoidea, then the vocal bands and all parts above them appear of a beautiful red color (of course the epiglottis is dark). But when we place the lamp near the cricoid cartilage, then we get a better survey of the whole subglottic region down to the bifurcation of the trachea, and this view is often more satisfactory than that obtained by the common method. But it must be observed that we have to become accustomed to this peculiar view of the larynx, it may be said, we see mostly in the negative. What, in ordinary illumination, impresses us as a thickening or an enlargement of a solid mass, in transillumination strikes us as a dark object. The rays of light cannot penetrate, and we infer that a solid mass, or a mass subject to the same optical laws, must intervene. We will therefore be able to define more precisely the contour of a tumor, because we see how the mass differentiates itself sharply by its dark outlines from the other parts in view. Only a few days ago I saw a patient of Dr. L. Weiss, who had a tumor on the posterior wall of the larynx, extending between the two cartilages from the left arytenoid downward. We can see the circumstance of it by means of transillumination better than by the ordinary method. I will have the pleasure of showing you the patient, and will be very glad to have the benefit of your opinion upon the case."

"Furthermore, we will be enabled to differentiate, in a certain case, between a solid tumor and a cyst, and this point seems to me to be of sufficient importance to give the method a fair trial. A few weeks ago I operated on a man who, a few minutes afterward, happened to be seen by Dr. Hoch, of Philadelphia. The patient had a tumor, the greatest part of which was below the glottis. It was attached to the right vocal band at the front angle, and extended over a little to the left vocal band. By means of

transillumination I could easily determine that it was a solid tumor, not a cyst, and could gauge the exact size of it. It is true that you could have excluded a cyst in this case even without transillumination, but there are cases where this is not so easily done, and for these I think transillumination is also of value."

"In this man, by the way, we could see, the day after the operation, that the edematous vocal band was much more translucent than before the edema had set in, and this brings us to the other class of cases, where we have a greater penetration of light than normally. This, however, can only be seen where loss of substance has taken place, as in ulcerative processes or in other pathological conditions which admit a greater penetration of light. When Gottstein[10] says, 'the reddened vocal band seems just as translucent as the normal,' he is correct; but you cannot draw from that a conclusion as to the usefulness of this method generally. Certainly we cannot recognize these changes, but there are others which we can see so much easier. So, for example, an edematous vocal band is differently translucent than an infiltrated one. The first is of a strong, light red, i. e., much redder than the normal, the last is much darker. This is a differentiation which is interesting enough to us."

While I have used transillumination in a few cases, my experience is too limited to warrant many positive conclusions, so I will, therefore, submit this test or method of examination for your approval in this "first communication," but I shall report more in detail later.

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## DISCUSSION.

**Dr. W. A. Walters**, Billings, Mont., said that to him, the field was new, he having had no experience with transillumination of the larynx by any method; but that he welcomed any new procedure of examination that offered any chance to gain substantial information or increase the accuracy of observation. He likened the present status of the subject to the early days of the X-ray and blood pressure investigations and expressed the hope that in time transillumination of the larynx would offer much of value. He agreed with the essayist that the differential diagnosis between cysts and other tumors of the larynx by transillumination could be readily accomplished.

**Dr. A. C. Magruder**, Colorado Springs, said: Recently when in the East, the subject was discussed in medical circles and many laryngologists looked with favor upon the method, believing there was much promise in it for satisfactory development. He encouraged Dr. Spencer to continue his efforts and observations with a view of publishing them in the future.

**Dr. T. E. Carmody**, Denver, congratulated Dr. Spencer on the work he had already done. He thought it a valuable procedure and one that offered many opportunities for development. He also referred to the differential diagnosis between cysts and other laryngeal tumors being greatly facilitated by transillumination.

**Dr. James Allen Patterson**, Colorado Springs, thanked Dr. Spencer for introducing such an interesting and new subject to the profession. He was pleased indeed that the paper was read at the Western Section Meeting as it contributed greatly to the general interest and success of the meeting. That the subject so full of promise should not be dropped without further investigation, was quite apparent and he with the others who had discussed the paper hoped to hear more about it from Dr. Spencer.

**Dr. F. R. Spencer**, Boulder, Colo., closing the discussion elaborated in detail the several methods of transillumination of the larynx.

# AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY.

## MINUTES.

The Twenty-third Annual Meeting of the American Laryngological, Rhinological and Otological Society convened at the Hotel Chelsea, Atlantic City, N. J., May 31st and June 1st, 1917.

The Society was called to order by the President, Dr. Thomas J. Harris, of New York City.

The following members registered during the sessions:

Dr. JOS. H. ABRAHAM.....New York
Dr. F. C. ARD.....Plainfield, N. J.
Dr. HUBERT ARROWSMITH.....Brooklyn, N. Y.
Dr. JAS. A. BABBITT.....Philadelphia, Pa.
Dr. JOHN F. BARNHILL.....Indianapolis, Ind.
Dr. JOSEPH C. BECK.....Chicago, Ill.
Dr. HUGH B. BLACKWELL.....New York
Dr. H. H. BRIGGS.....Asheville, N. C.
Dr. JOHN E. BROWN.....Columbus, O.
Dr. LOUIS J. BURNS.....Philadelphia, Pa.
Dr. RALPH BUTLER.....Philadelphia, Pa.
Dr. DON M. CAMPBELL.....Detroit, Mich.
Dr. WM. B. CHAMBERLIN.....Cleveland, O.
Dr. TALBOT R. CHAMBERS.....Jersey City, N. J.
Dr. GEO. M. COATES.....Philadelphia, Pa.
Dr. LEWIS A. COFFIN.....New York
Dr. LEE COHEN.....Baltimore, Md.
Dr. THEODORE CORWIN.....Newark, N. J.
Dr. GEO. F. COTT.....Buffalo, N. Y.
Dr. CHAS. N. COX.....Brooklyn, N. Y.
Dr. ROBERT H. CRAIG.....Montreal, Canada
Dr. JOHN F. CULP.....Harrisburg, Pa.
Dr. H. HOLBROOK CURTIS.....New York
Dr. FRANKLIN E. CUTLER.....Cleveland, O.
Dr. EWING W. DAY.....Pittsburgh, Pa.
Dr. LEE WALLACE DEAN.....Iowa City, Iowa
Dr. JESSE W. DOWNEY, Jr.....Baltimore, Md.
Dr. ARTHUR B. DUEL.....New York
Dr. JAMES GARFIELD DWYER.....New York
Dr. WELLS P. EAGLETON.....Newark, N. J.

Dr. THOMAS O. EDGAR.....Dixon, Ill.
Dr. FRANCIS P. EMERSON.....Boston, Mass.
Dr. LINN EMERSON.....Orange, N. J.
Dr. THOMAS H. FARRELL.....Utica, N. Y.
Dr. GEORGE FETTEROLF.....Philadelphia, Pa.
Dr. HENRY HALL FORBES.....New York
Dr. WOLF FREUDENTHAL.....New York
Dr. STANTON A. FRIEDBERG.....Chicago, Ill.
Dr. BEN CLARK GILE.....Philadelphia, Pa.
Dr. MAX A. GOLDSTEIN.....St. Louis, Mo.
Dr. CHARLES GRAEF.....New York
Dr. D. C. GREENE, Jr.....Boston, Mass.
Dr. J. H. GUNTZER.....New York
Dr. THOMAS H. HALSTED.....Syracuse, N. Y.
Dr. THOMAS J. HARRIS.....New York
Dr. WM. H. HASKIN.....New York
Dr. HILL HASTINGS.....Los Angeles, Cal.
Dr. CHRISTIAN R. HOLMES.....Cincinnati, O.
Dr. EDGAR M. HOLMES.....Boston, Mass.
Dr. HENRY HORN.....San Francisco, Cal.
Dr. JOHN HORN.....New York
Dr. HAMPTON P. HOWELL.....New York
Dr. E. V. HUBBARD.....New York
Dr. THOMAS HUBBARD.....Toledo, O.
Dr. LESTER MEAD HUBBY.....New York
Dr. LEE M. HURD.....New York
Dr. SAMUEL IGLAUER.....Cincinnati, O.
Dr. E. FLETCHER INGALS.....Chicago, Ill.
Dr. JOHN M. INGERSOLL.....Cleveland, O.
Dr. F. L. JACK.....Boston, Mass.
Dr. CHEVALIER JACKSON.....Philadelphia, Pa.
Dr. J. W. JERVEY.....Greenville, S. C.
Dr. WALTER B. JOHNSON.....Paterson, N. J.
Dr. RICHARD HALL JOHNSTON.....Baltimore, Md.
Dr. CLARENCE PORTER JONES.....Newport News, Va.
Dr. GEORGE F. KEIPER.....Lafayette, Ind.
Dr. BEVERLEY R. KENNON.....Norfolk, Va.
Dr. J. S. KIRKENDALL.....Ithaca, N. Y.
Dr. FRANK E. KITTREDGE.....Nashua, N. H.
Dr. DANIEL W. LAYMAN.....Indianapolis, Ind.
Dr. EUGENE R. LEWIS.....Dubuque, Iowa
Dr. FIELDING O. LEWIS.....Philadelphia, Pa.
Dr. ROBERT LEWIS.....New York
Dr. H. W. LOEB.....St. Louis, Mo.
Dr. JAS. E. LOGAN.....Kansas City, Mo.
Dr. STEPHEN H. LUTZ.....Brooklyn, N. Y.
Dr. HENRY LOWNDES LYNAH.....New York
Dr. ROBERT CLYDE LYNCH.....New Orleans, La.
Dr. GEORGE W. MacKENZIE.....Philadelphia, Pa.
Dr. D. MacPHERSON.....New York

Dr. PERCY E. D. MALCOLM.....New York
Dr. GEORGE PAULL MARQUIS.....Chicago, Ill.
Dr. WM. BEVERLY MASON.....Washington, D. C.
Dr. LEFFERTS A. McCLELLAND.....Brooklyn, N. Y.
Dr. O. A. M. McKIMMIE.....Washington, D. C.
Dr. RICHMOND McKINNEY.....Memphis, Tenn.
Dr. LEONIDAS L. MIAL.....New York
Dr. CLIFTON M. MILLER.....Richmond, Va.
Dr. ROBERT MILLIGAN.....Pittsburgh, Pa.
Dr. THOMAS W. MOORE.....Huntington, W. Va.
Dr. ROBERT L. MOOREHEAD.....Brooklyn, N. Y.
Dr. HARRIS P. MOSHER.....Boston, Mass.
Dr. GILBERT D. MURRAY.....Scranton, Pa.
Dr. ROBERT C. MYLES.....New York
Dr. JAMES F. McCAW.....Watertown, N. Y.
Dr. FRANCIS R. PACKARD.....Philadelphia, Pa.
Dr. JOHN R. PAGE.....New York
Dr. LAFAYETTE PAGE.....Indianapolis, Ind.
Dr. JAS. A. PATTERSON.....Colorado Springs, Colo.
Dr. WENDELL C. PHILLIPS.....New York
Dr. NORVAL H. PIERCE.....Chicago, Ill.
Dr. JOHN B. RAE.....New York
Dr. ANDREW J. N. REIK.....Baltimore, Md.
Dr. HENRY O. REIK.....Baltimore, Md.
Dr. GEORGE L. RICHARDS.....Fall River, Mass.
Dr. JOHN D. RICHARDS.....New York
Dr. CHARLES W. RICHARDSON.....Washington, D. C.
Dr. WALTER ROBERTS.....Philadelphia, Pa.
Dr. JOHN A. ROBINSON.....New York
Dr. SYLVAN ROSENHEIM.....Baltimore, Md.
Dr. GEORGE T. ROSS.....Montreal, Canada
Dr. DUNBAR ROY.....Atlanta, Ga.
Dr. C. C. SANDELS.....Pittsburgh, Pa.
Dr. RUFUS B. SCARLETT.....Trenton, N. J.
Dr. G. E. SHAMBAUGH.....Chicago, Ill.
Dr. ROSS HALL SKILLERN.....Philadelphia, Pa.
Dr. GREENFIELD SLUDER.....St. Louis, Mo.
Dr. HARMON SMITH.....New York
Dr. S. MacCUEN SMITH.....Philadelphia, Pa.
Dr. HERBERT E. SMYTH.....Bridgeport, Conn.
Dr. GEORGE E. STEEL.....New York
Dr. EDGAR J. STEIN.....Lancaster, Pa.
Dr. J. A. STUCKY.....Lexington, Ky.
Dr. C. A. THIGPEN.....Montgomery, Ala.
Dr. JOHN J. THOMSON.....Mt. Vernon, N. Y.
Dr. WALTER A. WELLS.....Washington, D. C.
Dr. JOSEPH A. WHITE.....Richmond, Va.
Dr. HAROLD WILSON.....Detroit, Mich.
Dr. D. J. GIBB WISHART.....Toronto, Canada

## REPORTS OF SECTION MEETINGS.

*Report of the Eastern Section.*

The annual meeting of the Eastern Section of this Society was held at the University Club in Philadelphia, Pa., on March 3d, 1917. The meeting which began promptly at 9:30 a. m. was very well attended. The morning program being devoted to the reading of six papers on ear, nose and throat subjects. The discussion of the papers was very free. At the conclusion of the morning program the Fellows of the Society and their guests had luncheon with the chairman of the section. The afternoon program which was purely clinical was held at the Jefferson College Hospital at 2:30 p. m. This clinical session was a very valuable part of the meeting, and there were ten separate demonstrations or operations shown or performed by prominent members of the Society or its guests. There were in attendance forty-nine members. The President of the Society Dr. Thomas J. Harris, of New York, and the dean of laryngology, Dr. J. Solis Cohen, of Philadelphia, honored the meeting by their presence. The chairman wishes to record his appreciation of the valuable assistance rendered by Dr. S. MacCuen Smith of Philadelphia in arranging for the clinical part of the program.

JOHN F. CULP,
Chairman of the Eastern Section.

*Report of the Southern Section.*

The meeting of the Southern Section was held in Memphis, Tennessee, on Saturday, March 24th, 1917. There were thirty members and guests present during the course of the meeting. At the morning session the following papers were read and discussed:—

Enucleation of the Faucial Tonsils by Guillotine Dissection—A Resume of Results. John J. Shea, M. D., Memphis, Tennessee.

A New Submucous Incision for Resection of the Nasal Septum. Clyde C. Purcell, M. D., Paducah, Kentucky.

A Clinical Report of Work in Bronchoesophagoscopy. R. H. T. Mann, M. D., Texarkana, Arkansas.

The Conservative Radical Mastoid (Heath) Operation—Its Indications, with Exhibition of Patients. P. M. Farrington, M. D., Memphis, Tennessee.

Lung Abscess as a Complication of Tonsillectomy. Ira Frank M. D., Chicago, Illinois.

At the afternoon session, held at the Memphis General Hospital, Dr. Joseph C. Beck, of Chicago, demonstrated the radical mastoid operation on two patients, Dr. R. C. Lynch, of New Orleans, Louisiana, operated for papilloma of the larynx, with suspension laryngoscopy, Dr. M. M. Cullom, of Nashville, Tennessee, demonstrated the technique of tonsillectomy, with instruments devised by him.

At the morning session, it was moved by Dr. Joseph C. Beck, seconded by Dr. R. C. Lynch, that the chairman of the section be requested to frame suitable resolutions of regret over the death of Dr. G. Hudson-Makuen, of Philadelphia, Pennsylvania.

A vote of thanks was extended to Dr. Richmond McKinney, chairman of the Southern Section, for having arranged for a very successfull meeting, and also for the hospitality extended by him to the visiting members and guests.

RICHMOND McKINNEY,
Chairman of the Southern Section.

*Report of the Middle Section.*

Doctor Brown in opening the meeting made a short address of welcome and announced the list of entertainments and clinics. He then introduced Dr. Thomas J. Harris, New York City, the national president of the Society who also gave us a short address and offered the following resolutions. Resolved, that one man from each state, comprising the Middle Section, form a committee to send condolence to the wife of Dr. Hudson-Makuen, who died during the week. The following committee was appointed. Drs. Thomas Hubbard, Ohio, chairman: Harold Wilson, Mich.; J. A. Stucky, Ken.; N. H. Pierce, Ill.; H. W. Loeb, Mo.; T. W. Moore, W. Va.; L. W. Dean, Iowa; D. W. Layman, Ind.; J. E. Brown, chairman and Thomas J. Harris, national president.

The visiting members of the Society were given the privilege of the floor. The first three papers by Doctor Hubbard "Functional and Organic Strictures of the Oesophagus." (Lantern Slides); Dr. J. W. Murphy "Some Experiences Demonstrating that Foreign Bodies in the Bronchi and Esophagus Are Dangerous"; Dr. S. Iglauer, "Removal of an Unusual Foreign Body from the Upper Esophagus, with Remarks on Preliminary Tracheotomy in Such Cases"; were read, and discussed by the following men: Doctors Beck, Thompson, Harris, Chamberlin, Loeb and Dean.

Dr. Samuel Iglauer also presented a paper, "Two Unusual Bronchoscopic Cases with Special Instruments Used in Extrac-

tion." After the discussion by Doctors Murphy, Hubbard, Chamberlin, Pierce, and Loeb the meeting adjourned to the Columbus Athletic Club for luncheon. The luncheon was tendered by the oto-laryngologists of Columbus.

The meeting was again called to order by Chairman Brown. Doctor Beck read his paper, "Further Report on the Malignant Disease of the Larynx by

(a) Percy coagulation method;
(b) Diathermia Coagulation method;
(c) Deep X-ray penetration;
(d) Massive radium application;
(e) Laryngectomy."

He then presented new instruments for holding the incised turbinals, clamp for holding adjustment made on fracture of the nose also electrodes for reducing hypertrophied tissues.

Doctor Iglauer presented an illuminator for the bronchoscope which he claims gives better illumination than any other style.

Dr. Thomas J. Harris then read his paper, "Dysphagia and Dysphonia of Dental Origin." (Case Reports). This was discussed by Doctors Ingersol, Thompson and Beck.

Dr. Clyde Brook's paper was next. "Some Phases of Drug Action of Interest to Oto-laryngologists." (By invitation). A vote of thanks was tendered him.

Dr. J. A. Stucky's paper, "The Serum Treatment of Fetid Coryza and Ozena" created a very spirited discussion, participated in by Doctors Harris, Senseney, Wilson, Chamberlin and Beck. Doctor Dean then presented his paper, "Report of a Case of Bilateral Congenital, Osseous Atresia of the External Auditory Canal with an Exceptionally Good Functional Result Following Operation," which was discussed by Doctors Pierce, Murphy, Beck and Chamberlin.

Dr. D. W. Layman closed the program by reading his paper, "Report of a Case of Acute Purulent Leptomeningitis of Otitic Origin."

The meeting adjourned to assemble again in the dining room where the chairman, Dr. John E. Brown gave a complimentary dinner to those present.

Those who remained were invited to the several hospitals for clinics the next morning. The meeting was a success in every way.

JOHN EDWARD BROWN,
Chairman of the Middle Section.

*Report of the Western Section.*

The chairman of the Western Section was requested by the President to do as much missionary work as possible. With the aid and advice of Dr. Levy about 1000 invitations were sent to all men known to be practicing laryngology exclusively in the states of Utah, Texas, Arizona, New Mexico, Kansas, Nebraska, Wyoming, Montana, Idaho and the Dakotas. Many replies were received. Quite a number of men expressed their desire to read papers of which they would give title later. A few desired to read papers on ophthalmological subjects which necessitated their refusal. The majority of those promising papers failed to furnish titles when repeatedly requested to do so.

The meeting however proved to be highly successful. The attendance was large, the papers were of a high degree of merit and were well discussed. The clinical feature aroused the greatest enthusiasm.

The success of the meeting was largely due to the loyalty of the fellows residing in the section and to the support of Drs. Dennis, Mullen, and Magruder, confreres practising laryngology in Colorado Springs.

J. A. PATTERSON, Secretary,
Western Section.

## EXECUTIVE SESSIONS.

May 31 to June 1, 1917.

*Election of Fellows:*

Seth A. Brumm, M. D., Philadelphia.
Ernest V. Hubbard, M. D., New York City.
George E. Shambaugh, Chicago.

*Appointing of Committees:*

Dr. J. A. Stucky, Dr. Charles W. Richardson, Dr. C. R. Holmes, Dr. T. M. Holmes, Dr. J. A. Patterson.

*Report of Secretary:*

To the Fellows of the American Laryngological, Rhinological and Otological Society:—

Gentlemen:—In submitting his first report the secretary first desires to thank all of the Fellows for their most kind consideration of his efforts throughout the past year for they have made his service one of much pleasure rather than a heavy burden.

On revising the list of Fellows the roll shows an active list of three hundred and two Fellows and twenty-two Honorary Fellows.

There has been three resignations during the past year, those of Dr. William Cheatham, Dr. Paul F. Sondern and Dr. Percy Fridenberg.

Three have been dropped for non-payment of dues.

The Society has lost four members by death, Dr. D. Braden Kyle, Dr. G. Hudson-Makuen, Dr. Amos Solenberger and Dr. H. S. Dye.

While the loss by death of any of our Fellows is always very deeply felt, this year has brought an almost irreparable loss to the Society in that two of the deceased Fellows were former presidents. No words of your secretary can express how great is the loss that the Society has sustained for their unfailing devotion and wise counsel in all important affairs affecting the welfare of the organization will be sorely missed by those who carry on the burdens from year to year.

It is deeply to be regretted that only two out of eleven theses submitted were considered by the Council to be of sufficient merit to warrant recommendation of the candidates for election. It is to be hoped that all proposers of candidates in the future will endeavor to make the candidates realize the necessity of making very careful preparation of their theses as their eligibility is largely determined by this evidence of their scientific ability. If this is done we can look forward to a very welcome increase

in our numbers as there are twenty seven applications now being considered by the Council.

It may be of interest to report that eighty Fellows and ninety-one guests have attended the several section meetings.

The Council has held two meetings during the past year, thirteen members attending the first meeting in January and twelve being present at the second. This clearly shows that the welfare of the Society has been considered of first importance by the men who have been honored by your choice for many of them have made real personal sacrifice to give their services in your behalf.

Respectfully submitted,
W. H. HASKIN, Secretary.

Moved and seconded that the Secretary's report be received and adopted. Carried.

The following resignations were presented by the Secretary:

Dr. Paul F. Sondern, New Jersey, Dr. W. Cheatham, Louisville, Ky., Dr. Percy Fridenberg, New York.

The question of the further division of the Society into five sections:

On motion, the motion proposed by Dr. Levy, of Denver, one year ago, was accepted.

The question of incorporation:

On motion, the recommendation of the Council that the Society be incorporated, was accepted.

*Report of the Treasurer*:

Pittsburgh, Pa., May 23d, 1917.

American Laryngological, Rhinological and Otological Society,
1005 Westinghouse Building. Pittsburgh, Pa.

Gentlemen:—

Acting under instruction from your Treasurer, Dr. E. W. Day, we have made an examination of the Cash Receipts and Disbursements, our report therefore does not show verification of Assets and Liabilities, we submit herewith two Exhibits as follows:—

Exhibit No. 1 Statement of Receipts and Disbursements, May 6, 1916, to May 19, 1917.

Exhibit No. 2. Statement of Investment Account.

The receipts of your Society, as set up in Exhibit No. 1, have been grouped by us under their different headings and all traced to your bank account, and all payments we found properly supported by vouchers. The balance on hand, as stated, was com-

pared with your bank checking account and found to be correct. The item of $20.00 appearing in your receipts as "Balance on Air Brake Exchange" was entered in the wrong account. This item should appear in your investment account. We would suggest that a voucher be drawn on your checking account for that amount and deposited to the credit of your investment account.

The investment securities owned by your Society and listed on Exhibit No. 2, were examined by our accountant in the presence of your Treasurer in the safe deposit vaults of the Commonwealth Trust Company of this City and found to be as stated. During the past year 40 shares of stock in the Union Switch and Signal Company were exchanged for 34 shares of Westinghouse Air Brake Company stock, your Society receiving Twenty ($20.00) Dollars in cash in addition.

The dividends and interest received from these securities have also been set up in Exhibit No. 2, together with the interest allowed by the bank on your cash balance. All these receipts have been traced by us to your bank "Savings Account", and the balance on hand, as stated, compared with your bank balance and found correct.

A list of your investments also appears on Exhibit No. 2, and the market value of the Pennsylvania R. R. stock together with that of the Westinghouse Air Brake set up as it was at the close of business, May 19th, 1917. We were unable to secure the market value of the Cresson Borough School Bonds so have entered them up at the same value as you carried them last year.

While the books of your Treasurer have been neatly and accurately kept, we feel that a great improvement could be made by the adoption of a Cash Book suitably ruled to meet your requirements. This would impose no extra work on your Treasurer and would give you, at any time, an accurate knowledge of the different sources from which your income was derived and at the same time would make a proper distribution of your expenses.

Respectfully submitted,

THE AUDIT COMPANY OF PITTSBURGH.
W. B. Scott. President.

Accountant in Charge:
J. E. Whittaker.

EXHIBIT NO. 1.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL & OTOLOGICAL SOCIETY STATEMENT OF RECEIPTS AND DISBURSEMENTS MAY 6 1916 TO MAY 19, 1917.

| | | |
|---|---|---|
| Balance May 6 1916 | | $1,474.99 |
| *Receipts.* | | |
| Transfers | $ 5.00 | |
| Dr. E. W. Day | 200.00 | |
| Do. | 296.00 | |
| Dues received year 1914-15 | 90.00 | |
| Dues received year 1915-16 | 390.00 | |
| Dues received year 1916-17 | 2,760.00 | |
| Initiation fees | 100.00 | |
| Balance received on Air Brake exchange | 20.00 | 3,861.00 |
| | | $5,335.99 |
| *Disbursements.* | | |
| Vol. No. | | |
| 1 Dr. R. C. Lynch | $ 50.00 | |
| 2 Andrew E. Loucks | 6.50 | |
| 3 Cash for meeting at Greenbrier Hill | 200.00 | |
| 4 Loy McAfee—Reporting meeting | 207.30 | |
| 5 Entertainment of ladies | 110.30 | |
| 6 Stamps | 5.00 | |
| 7 Programmes and envelopes | 86.50 | |
| 8 Lional Topaz Red Book | 3.00 | |
| 9 Wm. Chamberlin—Section meeting | 25.00 | |
| 10 Andrew E. Loucks—Letter heads, etc | 43.68 | |
| 11 Do.—Envelopes, etc. | 27.25 | |
| 12 Premium of treasurer's bond | 37.50 | |
| 13 Dr. E. W. Day—Stamps | 10.00 | |
| 14 Andrew E. Loucks—Envelopes | 3.75 | |
| 15 Safe deposit box | 5.00 | |
| 16 Dr. E. W. Day | 296.00 | |
| 17 Dr. T. J. Harris—Services | 25.00 | |
| 18 Murdock, Kerr Co.—Slips and blanks | 6.75 | |
| 19 Dr. Jas. Patterson—Expenses Western Section | 50.00 | |
| 20 Andrew E. Loucks—Letters and postage 23rd meeting | 25.50 | |
| 21 Dr. Carmody—Expenses section meeting | 25.00 | |
| 22 M. C. McGarvey—Filing and typewriting | 10.00 | |
| 23 Andrew E. Loucks—Proposals, papers and envelopes | 30.78 | |
| 24 T. J. Harris—Roses, $15.00; typewriting, $10.95 | 25.95 | |
| 25 Andrew E. Loucks—Printing | 23.00 | |
| 26 Dr. E. W. Day—Stamps | 10.00 | |
| 27 Mercury Pub. Co.—Transaction plates | 1,066.81 | |
| Postage, wrappers, etc | 84.33 | |
| 100 maps | 1.50 | |
| 28 Expenses of Secretary, 1916-17 | 250.00 | |
| 29 John F. Culp—Expenses Eastern Section meeting | 25.00 | |
| 30 Richmond McKinney—Expenses Southern meeting | 25.00 | |
| 31 G. L. Richards—Publication Committee | 53.57 | |
| 32 Andrew E. Loucks—Letters, envelopes, etc. | 20.00 | |
| 33 Dr. John E. Brown—Expenses meeting, Feb. 26 | 49.80 | |
| 34 Andrew E. Loucks—Printing | 7.25 | |
| | | 2,932.02 |
| Balance on hand in checking account | | $2,403.97 |

EXHIBIT NO. 2.

STATEMENT OF INVESTMENT ACCOUNTS AT THE CLOSE OF BUSINESS MAY 17, 1917.

*Investments.*

| | Par Value | Market Value |
|---|---|---|
| 100 shares Penna. R. R. stock | $ 5,000.00 | $ 5,262.50 |
| 34 shares Westinghouse Air Brake stock | 1,700.00 | 3,893.00 |
| 8 bonds Cresson Borough School Bonds ($500) | 4,000.00 | 4,037.35 |
| | $10,700.00 | $13,192.85 |

Cash deposited with People's National Bank, Pittsburgh, Pa., in Savings Account, pass book No. F127, as follows:—

| | | | | |
|---|---|---|---|---|
| April 26, 1916. | Balance | | | $1,004.42 |
| May 3, 1916. | 1½ % | dividend P. R. R. stock | $75.00 | |
| July 10, 1916. | 2¼ % | Int. Cresson School bonds | 90.00 | |
| July 17, 1916. | 1½ % | dividend Union S. & S. Co. | 60.00 | |
| Sept. 5, 1916. | 1½ % | dividend P. R. R. stock | 75.00 | |
| Oct. 14, 1916. | 1½ % | dividend Union S. & S. Co. | 60.00 | |
| Dec. 1, 1916. | | Interest on savings | 25.01 | |
| Dec. 1, 1916. | 1½ % | dividend P. R. R. stock | 75.00 | |
| June 1, 1915. | | Interest on savings | 17.78 | |
| Dec. 20, 1916. | 2¼ % | Int. Cresson School bonds | 90.00 | |
| Jan. 20, 1917. | 1½ % | dividend Union S. & S. Co. | 60.00 | |
| April 7, 1917. | 1½ % | dividend P. R. R. stock | 75.00 | |
| April 23, 1917. | 1¾ % | dividend Westinghouse Air Brake | 59.50 | |
| | | | | 762.29 |
| | | Balance as stated in Savings Account pass book | | $1,766.71 |

Moved and seconded that the report of the Treasurer be received and adopted. Carried.

*Report of the Librarian*:

The question of the disposition of the Library of the Society was presented by the President who stated that the virtual retirement from practice of Dr. H. Holbrook Curtis, who, as Chairman of the Library Committee and Librarian, had collected and housed the Library, made it necessary to arrange for other disposition of the collection of reprints and other pamphlets. The Council had communicated with the New York Academy of Medicine, with the result that that institution had expressed a willingness to take over the Library of the Society, and to give it a special place, duly catalogued in the Library of the Academy.

On motion, the offer of the New York Academy of Medicine to take charge of the Library of the Society was accepted.

On motion, a vote of thanks was tendered Dr. Curtis for his untiring efforts in connection with the Library Committee.

*Report of the Committee on Nominations*:

President—Dr. George L. Richards, Fall River, Mass.

Vice-Presidents—Eastern Section, Dr. D. Crosby Greene, Jr., Boston, Mass. Middle Section, Dr. D. W. Layman, Indianapolis, Ind. Southern Section, Dr. Thomas W. Moore, Huntington, W. Va. Mid-Western Section, Dr. Frank R. Spencer, Boulder, Colo. Western Section, Dr. H. B. Graham, San Francisco, Cal.

Secretary—Dr. William Henry Haskin, New York City.

Treasurer—Dr. Ewing W. Day, Pittsburgh, Pa.

Members of Council—Dr. T. J. Harris, New York City. Dr. Francis P. Emerson, Boston, Mass., Dr. L. W. Dean, Iowa City, Ia.

Publication Committee—Dr. O. H. Jackson, Fall River, Mass., Dr. E. M. Holmes, Boston, Mass., Dr. Arthur I. Weil, New Orleans, La.

It was moved and seconded that the report be received and adopted. Carried.

## GEO. HUDSON-MAKUEN.

George Hudson-Makuen was born in Goshen, N. Y., July 16, 1855, and died in the same city February 21, 1917. The foundation for his life work was well laid, Yale University (1884) being his Alma Mater in arts, and Jefferson Medical College (1889) in medicine. After several years in general medicine in Philadelphia he entered upon the specialty of laryngology and otology, and soon attained a prominent position in this department of medicine.

For more than twenty years it was our privilege to know him with increasing intimacy, and the sincerity of his friendship served to bind us more closely to him with each succeeding year. We leave it to others to tell the story of his professional achievements, of the distinguished and in many ways unique position that he held in the Causes and Treatment of Defects of Speech, in which it can with truth be said that he stood alone. We desire only to emphasize those personal qualities which came out as we were thrown with him in the affairs of this Society.

He became a member at its organization in 1896. He was Vice-President and Chairman of the Eastern Section in 1900, President from 1912-13, member of its Council for four years. In all these relations he discharged his duties with faithfulness

and ability. His was a gentle nature. Tenacious of his views, he always advanced them with courtesy and with unfailing consideration of others. We never heard him say an unkind thing of anyone nor knew him willingly to wound another's feelings. As an after-dinner speaker he was especially gifted and never was at a loss for a story to enliven what he was saying. Many of us here recall with lasting pleasure the cordial hospitality which we experienced at the hands of his lifelong companion and himself when we visited them in their Chester home at the time that he was President of the Society.

To her in her great sorrow we extend our deep sympathy. As for our friend, our faith is such that we can say of him, as the poet did of his friend, Arthur Hallam, "I count it truth with him who sings to one clear harp of divers tones that men may rise on stepping-stones of their dead selves to higher things."

## DAVID BRADEN KYLE.

It is with deep regret and real sorrow that we record the untimely death, on October 23, 1916, of our dear friend and colleague, David Braden Kyle, of Philadelphia, an event deeply deplored not only by the Fellows of this Society, but by his hosts of friends and admirers.

Dr. Kyle was born in Cadiz, O., in 1863. He received his education in the public schools and Muskingum College, and was graduated from the Jefferson Medical College of Philadelphia in 1891. During his collegiate course Dr. Kyle developed special aptitude for bacteriology and pathology, and in these two branches he probably excelled among American laryngologists, as is shown by his writings and his Text Book of Diseases of the Nose and Throat, now in its fifth edition. In recognition of his attainments in pathology, he was selected to teach this branch in the Jefferson Medical College during an interim year. Following this, he was elected to the Chair of Laryngology at Jefferson, which position he held at the time of his death.

Dr. Kyle possessed a remarkably persuasive and pleasing personality, which served him well in his earlier life as a teacher in the public schools of his native town, and this work, incidentally, laid the foundation for his future successful and distinguished career as a teacher, invention and development of instruments and appliances for use in his specialty.

Dr. Kyle was accorded the unusual honor of being twice elected to the presidency of the American Laryngological Society. He also served a term as president of this Society.

Dr. Kyle was able to impart much of his own enthusiasm for his chosen work to the hundreds of students whom he taught, and to this large body of professional men his death has come as a great shock and a personal loss.

Those of us who have known Dr. and Mrs. Kyle and appreciated the rare comradeship and real devotion that have always been such a notable feature in their life must extend to her our heartfelt sympathy in her irreparable loss.

## AMOS R. SOLENBERGER.

Dr. Solenberger became a member of the Society in 1901 and served as Vice-President and Chairman of the Western Section in 1910. He occupied an enviable position in the city of his adoption where his untimely departure is greatly deplored.

## HOBART S. DYE.

Dr. Dye became a member of the Society in 1902. He was regular in his attendance at the meetings of the Society. Distinguished in appearance, kindly in manner, faithful in his profession, he stood for all that characterizes the true gentleman. All who knew him will deplore his departure from our midst.

S. MacCuen Smith,
Arthur B. Duel,
Thomas J. Harris.

## Report of the Committee Representing The American Otological Society, The American Laryngological Association, and The American Laryngological, Rhinological and Otological Society to The Council of National Defense.

Having been asked by the Council of National Defense to suggest in which way the nose, throat and ear specialists of the societies mentioned can be of use to the Army and Navy Medical Corps, we feel that we can suggest the following:

We recommend that all members of the American Otological Society, the American Laryngological Association, and the American Laryngological, Rhinological and Otological Society be requested to become members of the Medical Reserve Corps of the Army and Navy, or of the Officers Reserve.

In the first place, the members of these societies could examine recruits for the aviation service, in testing the condition of their labyrinths.

In the second place, we could be of service in examining the upper air tract of recruits, especially in cases of hoarseness or other suspicious conditions which might evidence the possibility of tuberculosis.

In the third place, we could be of service in examining all recruits in whom there was a suspicion of impairment of hearing, or in which there was any evidence or suspicion of the infection of the middle ear, either acute or chronic. We recognize the importance of excluding chronic suppurative ears, from the experience of the British Army.

We could be of service in the organization of a base hospital for the treatment of cases of injuries and diseases of the eye, ear, nose and throat at the base of military operations or elsewhere.

The younger man or junior is the physician under thirty-five years of age, and the consultant or senior is the physician over thirty-five years of age.

It is advised that the younger men be utilized for active service in the base or other hospitals, and that the older men be utilized as consultants for one or more base hospitals, or a group of neighboring hospitals.

It is advised that a list of the Naval and Base Hospitals, and Naval Training Stations be obtained, and a list made of the consulting specialists. This list to be placed in the hands of the Army and Navy officials in order that in case of need the required specialists may be assigned to the various hospitals. The names of these specialists are to be chosen by a committee from the three special societies represented by this committee.

We should like to call attention to the necessity of testing the hearing of men in military service in order to ascertain the type of impairment of hearing that will exclude men from certain forms of service, yet permit them to engage in other forms; and to formulate and publish these tests for the use of the consulting military throat and ear specialists of the country.

We recommend that men furloughed from the front, having affections of the nose, throat and ear, be referred to some distinct hospital and some designated physician in connection with that hospital, in order that the physician may keep in touch with the soldier or sailor until he is sent back to the line of service.

CHARLES W. RICHARDSON,
*Representing the*
*American Otological Society.*
BURT R. SHURLEY,
*Representing the*
*American Laryngological Association.*
HARRIS P. MOSHER,
*Representing the*
*American Laryngological, Rhinological and Otological Society.*

It was moved and seconded that the report of the Committee of National Defense be accepted and placed on file. Carried.

It was moved and seconded that the Committee be continued. Carried.

The following motion, proposed by Dr. Ewing W. Day, was seconded and carried:

That the Committee of National Defense be empowered to consult with the Surgeon General of the Army and Navy: that it be suggested that Medical Officers of the Reserve Corps be employed, as far as possible, in line with their respective specialties; that it be suggested that one or more base hospitals for diseases of the ear, nose and throat be established; that the Committee be empowered to take up any questions which may arise in connection therewith.

It was moved and seconded that the Committee of National Defense be instructed to go before the Sections of Oto-Laryngology and Ophthalmology of the American Medical Association at its Annual Meeting in New York City. Carried.

The following recommendation of the Council, arising out of the crisis of the war, was presented by Dr. Day:

That, in view of what may be expected for the next two or three years, it is thought best to suspend dues for all members actively in service; also to cut off all unnecessary expenses, including the Section meetings and numerous other incidentals; and that members who go into service shall notify the Treasurer's office.

Moved that the recommendation of the Council with reference to the suspension of the section meetings be accepted.

Proposed amendment to the motion: That it be left to the in-coming administration to determine whether or not section meetings are advisable.

Further amendment: That all details, with reference to section meetings and other business, be left to the in-coming administration.

Question: That the in-coming Council shall have full power with reference to section meetings and all business matters for the coming year. Seconded. Carried.

The President announced that through the splendid efforts of Dr. Emil Mayer, of New York City, in behalf of the widow of Dr. Jules Broeckaert, the Belgian otologist, several thousand dollars had been raised for her, thus, with what had been done in England, placing her beyond want.

The question of the publication, by the American Medical Association, of a journal devoted to Oto-Laryngology, was presented by Dr. E. Fletcher Ingals.

On motion, the communication presented by Dr. Ingals was referred to the Council.

Adjourned.

## Executive Session, Friday Afternoon.

Election of Officers:

On motion, the Secretary was instructed to cast the ballot of the Society for the list of officers as presented by the Committee on Nominations.

## FELLOWS.

Abraham, Jos. H. ..... 130 West 58th St., New York
Abbott, Wm. J. ..... 210 Lennox Bldg., Cleveland, Ohio
Alexander, Lawrence Dade ..... 130 West 59th St., New York
Allen, John H. ..... 711 Congress St., Portland, Me.
Allport, Frank ..... 72 E. Madison St., Chicago, Ill.
Ard, F. C. ..... 604 Park Ave., Plainfield, N. J.
Arrowsmith, Hubert ..... 170 Clinton St., Brooklyn, N. Y.
Atkinson, Wm. J. ..... 111 North Main St., Paterson, N. J.
Babbitt, Jas. A. ..... 1901 Chestnut St., Philadelphia, Pa.
Bane, Wm. C. ..... Metropolitan Bldg., Denver, Colo.
Barndollar, Wm. P. ..... Westinghouse Bldg., Pittsburgh, Pa.
Barnhill, John F. ..... Pennway Bldg., Indianapolis, Ind.
Barnes, Harry A. ..... 213 Beacon St., Boston, Mass.
Beck, Joseph C. ..... 2551 N. Clark St., Chicago, Ill.
Berens, T. Passmore ..... 2041 Chestnut St., Philadelphia, Pa.
Bird, Urban Sinclair ..... Tampa, Fla.
Birkett, H. S. ..... 252 Mountain St., Montreal, Can.
Blackwell, Hugh B. ..... 148 W. 58th St., New York
Blake, Clarence John ..... 226 Marlboro St., Boston, Mass.
Borden, Charles R. C. ..... 484 Beacon St., Boston, Mass.
Braislin, W. C. ..... 425 Clinton Ave., Brooklyn, N. Y.
Braun, Alfred ..... 616 Madison Ave., New York
Briggs, H. H. ..... 73 Haywood St., Asheville, N. C.
Brown, Clayton M. ..... 158 North Pearl St., Buffalo, N. Y.
Brown, H. Beattie ..... 53 West 52nd St., N. Y. City
Brown, John E. ..... 239 E. Town St., Columbus, Ohio
Brown, John Mackenzie, 1002 Brockman Bldg., Los Angeles, Cal.
Brown, J. Price ..... Cranbrook Lodge Wycombe, Ont., Can.
Brumm, Seth A. ..... 4813 Baltimore Ave., Philadelphia, Pa.
Bryant, W. Sohier ..... 19 W. 54th St., New York
Burns, Louis J. ..... 1906 Chestnut St., Philadelphia, Pa.
Butler, Ralph ..... 1926 Chestnut St., Philadelphia, Pa.
Campbell, Don M. ..... 801 Peter Smith Bldg., Detroit, Mich.
Campbell, Wm. E. ..... 606 Century Bldg., Atlanta, Ga.
Canfield, R. Bishop ..... Ann Arbor, Mich.

Carmody, Thos. E.....Metropolitan Bldg., Denver, Colo.
Carter, Wm. Wesley.....69 W. 50th St., New York
Chamberlin, Wm. B.....1021 Prospect Ave., Cleveland, Ohio
Chambers, Talbot R.....15 Exchange Pl., Jersey City, N. J.
Chappell, Walter F.....7 E. 55th St., New York
Chenery, Wm. E.....222 Huntington Ave., Boston, Mass.
Chisholm, W. Alexander.....68 W. 55th St., New York
Cline, L. C.....224 North Meridian St., Indianapolis, Ind.
Coates, Geo. M.....1736 Pine St., Philadelphia, Pa.
Cobb, Frederick C.....11 Marlboro St., Boston, Mass.
Cocks, Gerhard H.....137 E. 54th St., New York
Coffin, Lewis A.....616 Madison Ave., New York
Coggeshall, Henry.....40 E. 58th St., New York
Cohen, Lee.....1820 Eutaw Place, Baltimore, Md.
Cohen, J. Solis.....2113 Chestnut St., Philadelphia, Pa.
Collins, Burnett.....645 St. Marks Ave., Brooklyn, N. Y.
Connor, Ray.....503 Washington Arcade, Detroit, Mich.
Corwin, Theodore.....20 Central Ave., Newark, N. J.
Coolidge, Algernon.....613 Beacon St., Boston, Mass.
Cooper, Claude E.....Metropolitan Bldg., Denver, Colo.
Cott, Geo. F.....1195 Main St., Buffalo, N. Y.
Cox, Chas. N.....257 Jefferson Ave., Brooklyn, N. Y.
Craig, Robert H.....510 Sherbrooke St., W., Montreal, Can.
Crockett, Eugene A.....298 Marlboro St., Boston, Mass.
Culbert, Wm. Ledlie.....16 E. 54th St., New York
Culp, John F.....410 No. Third St., Harrisburg, Pa.
Curtis, H. Holbrook.....853 Seventh Ave., New York
Cutler, Franklin E.....936 Rose Bldg., Cleveland, Ohio
Davis, George E.....50 W. 37th St., New York
Day, Ewing W.....Westinghouse Bldg., Pittsburgh, Pa.
Daly, Walter S.....Ogdensburg, N. Y.
Dean, Lee Wallace.....Iowa City, Iowa
Demarest, Frederick F. C.....29 Academy St., Passaic, N. J.
Deneh, Edward B.....15 E. 53rd St., New York
Downey, Jesse W., Jr.....529 N. Charles St., Baltimore, Md.
Dudley, Wm. H.....512 Brockman Bldg., Los Angeles, Cal.
Duel, Arthur B.....27 E. 57th St., New York
Dunn, John.....411 East Franklin St., Richmond, Va.
Durkee, John W.....142 Clinton St., Brooklyn, N. Y.
Dwyer, James Garfield.....40 E. 41st St., New York
Eagleton, Wells P.....15 Lombardy St., Newark, N. J.
Edgar, Thomas O.....Dixon National Bank Bldg., Dixon, Ill.

Ellegood, Joshua A.....9th and Market Sts., Wilmington, Del.
Ellis, H. Bert.....245 Bradbury Bldg., Los Angeles, Cal.
Emerson, Francis P.....520 Commonwealth Ave., Boston, Mass.
Emerson, Linn.....Metropolitan Bldg., Orange, N. J.
Eves, Curtis C.....1700 Walnut St., Philadelphia, Pa.
Fairbairn, John F.....131 Allen St., Buffalo, N. Y.
Farrell, Thomas H.....236 Genessee St., Utica, N. Y.
Fetterolf, George.....134 South 20th St., Philadelphia, Pa.
Fisher, Mark O.....Parkersburg, W. Va.
Fisher, Stanwood E.....190 State St., Portland, Me.
Fitzpatrick, T. V.....26 Garfield Place, Cincinnati, Ohio
Fleming, Ernest W..Trust & Savings Bldgs., 6th and Spring Sts., Los Angeles, Cal.
Forbes, Henry Hall.....40th E. 41st St., New York
Forsyth, Edgar A.....Metropolitan Bldg., Buffalo, N. Y.
Foster, John M.....11 Stedman Bldg., Denver, Colo.
Fowler, Edmond P.....616 Madison Ave., New York
Frank, Ira.....104 So. Michigan Ave., Chicago, Ill.
Freudenthal, Wolf.....59 East 75th St., New York
Friedberg, Stanton A.....104 So. Michigan Ave., Chicago, Ill.
Friedenwald, Harry.....1029 Madison Ave., Baltimore, Md
Friesner, Isidore.....814 Lexington Ave., New York
Gallaher, Thomas J.....California Bldg., Denver, Colo.
Gile, Ben Clark.....1906 Chestnut St., Philadelphia, Pa.
Goldsmith, Perry G.....84 Carlton St., Toronto, Can.
Goldstein, Max A.....3858 Westminster Place, St. Louis, Mo.
Graef, Charles.....1125 Boston Road, New York
Graham, Harrington Bidwell, 240 Stockton St., San Francisco, Cal.
Grayson, Charles P.....262 South 15th St., Philadelphia, Pa.
Greene, D. C., Jr.....483 Beacon St., Boston, Mass.
Greene, Jos. Berry.....Medicine Bldg., Asheville, N. C.
Guggenheim, Louis K.....1006 Carleton Bldg., St. Louis, Mo.
Guntzer, J. H.....40 East 41st St., New York
Griffin, E. Harrison.....55 West 47th St., New York
Halsted, Thomas H.....831 University Block, Syracuse, N. Y.
Harris, Thomas J.....104 East 40th St., New York
Hartz, Henry J.....619 Whitney Bldg., Detroit, Mich.
Haskin, Wm. H.....West Point, N. Y.
Hastings, Hill.....Trust & Savings Bldgs., 6th and Spring Sts.. Los Angeles, Cal.
Hays, Harold.....11 W. 81st St., New York
Hedges, Halstead S.....Charlottesville, Va.

Held, R. Johnson.....616 Madison Ave., New York
Hinkel, F. W.....581 Delaware Ave., Buffalo, N. Y.
Holmes, Christian R.....8 East 8th St., Cincinnati, Ohio
Holmes, Edgar M.....531 Beacon St., Boston, Mass.
Hopkins, F. E.....25 Harrison Ave., Springfield, Mass.
Horn, Henry.....209 Post St., San Francisco, Cal.
Horn, John.....72 East 92nd St., New York
Howell, Hampton P.....616 Madison Ave., New York
Hubbard, E. V.....17 East 38th St., New York
Hubbard, Thomas.....515 Nicholas Bldg., Toledo, Ohio
Hubby, Lester Mead.....27 West 68th St., New York
Hurd, Lee M.....15 East 48th St., New York
Hyatt, Frank.....The Rochambeau, 815 Connecticut Ave., Washington, D. C.
Iglauer, Samuel.....7th and Race Sts., Cincinnati, Ohio
Imperatori, Charles J.....17 East 38th St., New York
Ingals, E. Fletcher.....104 South Michigan Ave., Chicago, Ill.
Ingersoll, John M.....1021 Prospect Ave., Cleveland, Ohio
Jack, F. L.....215 Beacon St., Boston, Mass.
Jackson, Chevalier.....Walnut and 17th St., Philadelphia, Pa.
Jackson, Oliver H.....34 North Main St., Fall River, Mass.
Jervey, J. W.....Greenville, S. C.
Joachim, Otto.....501 Macheca Bldg., New Orleans, La.
Johnson, Walter B.....170 Broadway, Paterson, N. J.
Johnston, Richard Hall.....807 N. Charles St., Baltimore, Md.
Jones, Clarence Porter.....3117 West Ave., Newport News, Va.
Keiper, George F.....Lafayette, Ind.
Kenefick, Jos. A.....103 East 55th St., New York
Kennon, Beverley R.....64 Granby St., Norfolk, Va.
Kerrison, Philip D.....58 West 56th St., New York
Kirkendall, J. S.....Ithaca, N. Y.
Kittredge, Frank E.....Masonic Temple, Nashua, N. H.
Knowles, W. F.....220 Clarendon St., Boston, Mass.
Kopetzky, Samuel J.....616 Madison Ave., New York
Kuyk, D. A.....114 North 5th St., Richmond, Va.
Kyle, John J.....Title Insurance Bldg., Los Angeles, Cal.
Large, Secord H.....536 Rose Building, Cleveland, Ohio
Lasalle, J. J.....226 Michigan St., Toledo, Ohio
Layman, Daniel W.....608 Hume-Mansur Bldg., Indianapolis, Ind.
Lederman, M. D.....58 East 75th St., New York
Leland, George A.....354 Commonwealth Ave., Boston, Mass.
Lester, John C.....432 Madison Ave., New York

Levy, Robert.....Metropolitan Building, Denver, Colo.
Lewis, Eugene R.....1130 Main St., Dubuque, Iowa
Lewis, Fielding O.....261 So. 17th St., Philadelphia, Pa.
Lewis, Robert.....48 West 40th St., New York
Lincoln, Wm. R.....Lenox Bldg., Cleveland, Ohio
Linhart, C. P.....106 East Broad St., Columbus, Ohio
Lockard, Lorenzo B.....Metropolitan Bldg., Denver, Colo.
Loeb, H. W.....537 North Grand Ave., St. Louis, Mo.
Logan, Jas. E.....1208 Wyandotte St., Kansas City, Mo.
Loughran, Robt. Livingston.....46 West 52nd St., N. Y.
Lowman, John B.....142 Park Place, Johnstown, Pa.
Lutz, Stephen H.....284 Hancock St., Brooklyn, N. Y.
Lynah, Henry Lowndes.....127 W. 58th St., New York
Lynch, Robert Clyde, 634 Maison Blanche Bldg., New Orleans, La.
MacKenty, John Edmund.....43 West 54th St., New York
MacKenzie, George W.....1831 Chestnut St., Philadelphia, Pa.
MacPherson, D.....616 Madison Ave., New York
Malcolm, Percy E. D.....410 Park Ave., New York
Martinez, Emilio.....56 Neptune St., Havana, Cuba
Marquis, George Paull.....30 N. Michigan Ave., Chicago, Ill.
Mason, Wm. Beverly.....1738 M St., Washington, D. C.
Matthews, Justus.....1021 Metropolitan Bank Bldg., Minneapolis, Minn.
Means, Chas. S.....131 East State St., Columbus, Ohio
Menge, Frederick.....25 East Washington St., Chicago, Ill.
Mercer, Wm. F.....400 West Grace St., Richmond, Va.
Mial, Leonidas L.....40 East 41st St., New York
Miller, Clifton M.....217 East Grace St., Richmond, Va.
Miller, Frank E.....17 West 54th St., New York
Miller, H. Edward.....310 Metropolitan Bldg., St. Louis, Mo.
Milligan, Robert.....Westinghouse Bldg., Pittsburgh, Pa.
Mooney, Jas. J.....490 Delaware Ave., Buffalo, N. Y.
Moore, Thomas W., .1st National Bank Bldg., Huntington, W. Va.
Moorhead, Robt. L.....185 Hancock St., Brooklyn, N. Y.
Morgenthau, George.....104 S. Michigan Blvd., Chicago, Ill.
Mosher, Harris P.....828 Beacon St., Boston, Mass.
Moss, Robert E.....Hicks Bldg., San Antonio, Tex.
Munger, Carl E.....Waterbury, Conn.
Murphy, John W. .2700 Union Central Life Bldg., Cincinnati, Ohio
Murray, Gilbert D.....516 Spruce St., Scranton, Pa.
Myers, Harry L.....Taylor Bldg., 151 Granby St., Norfolk, Va.
Myles, Robert C.....11 E. 48th St., New York

McCaw, James F.....114 Sterling St., Watertown, N. Y.
McClelland, Lefferts A.....78 McDonough St., Brooklyn, N. Y.
McCoy, John J.....157 West 73rd St., New York
McCready, Jas. Homer.....816 Empire Bldg., Pittsburgh, Pa.
McCullagh, Samuel.....108 W. 58th St., New York
McKernon, Jas. F.....62 West 52nd St., New York
McKimmie, O. A. M.....1330 Mass. Ave., N. W. Washington, D. C.
McKinney, Richmond.....Bank of Commerce and Trust Bldg., Memphis, Tenn.
McLaury, Frank H.....Westport, Conn.
McReynolds, John O.....Trust Bldg., Dallas, Tex.
Nardin, Walter H.....Bleckley Bldg., Anderson, S. C.
Oertel, Theodore E.....Augusta, Ga.
Off, Henry J.....115 South 20th St., Philadelphia, Pa.
O'Kelley, Jas. P.....501 Macheca Bldg., New Orleans, La.
Oppenheimer, Seymour.....45 East 60th St., New York
Packard, Francis R.....304 South 19th St., Philadelphia, Pa.
Page, John R.....127 East 62nd, St., New York
Page, Lafayette.....224 N. Meridian St., Indianapolis, Ind.
Park, J. Walter.....32 North 2nd St., Harrisburg, Pa.
Parker, Edward F.....70 Hassel St., Charleston, S. C.
Patterson, Jas. A.....214 Burns Bldg., Colo. Springs, Colo.
Phillips, Wendell C.....40 West 47th St., New York
Pierce, Norval H.....22 E. Washington St., Chicago, Ill.
Pischell, Kaspar.....135 Stockton St., San Francisco, Cal.
Rae, John B.....247 West 70th St., New York
Ray, J. M.....432 West Chestnut St., Louisville, Ky.
Reik, Andrew J. N.....506 Cathedral St., Baltimore, Md.
Reik, Henry O.....506 Cathedral St., Baltimore, Md.
Renner, W. Scott.....341 Linwood Ave., Buffalo, N. Y.
Richards, George L.....124 Franklin St., Fall River, Mass.
Richards, John D.....8 East 54th St., New York
Richardson, Charles W., 1317 Connecticut Ave., Washington, D. C.
Roberts, Walter.....33 South 19th St., Philadelphia, Pa.
Roberts, W. Humes.....461 East Colo. St., Pasadena, Cal.
Robinson, John A.....40 East 41st St., New York
Root, Arthur G.....218 States St., Albany, N. Y.
Rosenheim, Sylvan.....1710 Linden Ave., Baltimore, Md.
Ross, George T.....456 Mackay St., Montreal, Can.
Roy, Dunbar.....Grand Opera House Block, Atlanta, Ga.
Sandels, C. C.....Westinghouse Bldg., Pittsburgh, Pa.
Sauer, Wm. E.....Grand and Washington Aves., St. Louis, Mo.

Scarlett, Rufus B.....78 North Clinton Ave., Trenton, N. J.
Scruton, William A.....27 E. 57th St., New York
Senseney, Eugene T.....308 Lister Bldg., St. Louis, Mo.
Shambaugh, Geo. E.....People's Gas Bldg., Chicago, Ill.
Sharp, J. Clarence.....62 West 46th St., New York
Shattuck, Warren S.....160 Clinton St., Brooklyn, N. Y.
Shorter, J. H.....Macon, Ga.
Shurly, B. R.....32 West Adams Ave., Detroit, Mich.
Simpson, John R.....Westinghouse Bldg., Pittsburgh, Pa.
Skillern, Ross Hall.....2032 Chestnut St., Phila., Pa.
Sluder, Greenfield.....3542 Washington Ave., St. Louis, Mo.
Smith, A. John.....Yonkers, New York
Smith, Harmon.....44 West 49th St., New York
Smith, Owen.....692 Congress St., Portland, Me.
Smith, S. MacCuen.....1429 Spruce St., Philadelphia, Pa.
Smith, W. Harvey.....173 Portage Ave. E., Winnipeg, Manit.
Smith, E. Terry.....75 Pratt St., Hartford, Conn.
Smyth, Herbert E.....376 John St., Bridgeport, Conn.
Spencer, Frank R.....Physicians' Block, Boulder, Colo.
Sperry, Fred'k N.....33 College St., New Haven, Conn.
Spratt, Charles N.....Reid, cor 9th St. and Nicollet Ave. Minneapolis, Minn.
Steel, George E.....256 W. 79th St., New York
Stein, Edgar J.....234 North Duke St., Lancaster, Pa.
Stein, Otto Jacob.....30 North Michigan Ave., Chicago, Ill.
Stickle, Charles Waldo.....86 Pierrepont St., Brooklyn, N. Y.
Stillman, F. L.....118 East Broad St., Columbus, Ohio
Stout, George C.....1611 Walnut St., Philadelphia, Pa.
Stucky, J. A.....The McClelland Bldg., Lexington, Ky.
Sullivan, Jno. Jas., Jr.....401 Traders Bank Bldg., Scranton, Pa.
Theisen, Clement F.....172 Washington Ave., Albany, N. Y.
Thigpen, C. A.....13 South Ferry St., Montgomery, Ala.
Thigpen, F. M.....13 South Ferry St., Montgomery, Ala.
Thompson, John A.....628 Elm St., Cincinnati, Ohio
Thomson, John J.....3 Park Ave., Mt. Vernon, N. Y.
Thrasher, A. B.....N. E. Cor. 7th and Race Sts., Cincinnati, Ohio
Thrasher, Wade....N. E. Cor. 7th and Race Sts., Cincinnati, Ohio
Trowbridge, D. H.....Fresno, Cal.
Tunis, Jos. P..The Gladstone, 11th and Pine Sts., Philadelphia, Pa.
Turnbull, Thomas.....835 Western Ave., Pittsburgh, Pa.
Vail, Derrick T.....24 East 8th St., Cincinnati, Ohio

Vanderhoof, Don Allison.....Exchange National Bank Bldg. Colorado Springs, Colo.
Vansant, Eugene L.....1929 Chestnut St., Philadelphia, Pa.
Voislawsky, A. P.....128 West 59th St., New York
Voorhees, Irving Wilson..14 Central Park West, New York, N. Y.
Wagner, Henry L.....518 Sutter St., San Francisco, Cal.
Wales, Ernest DeW...1336 N. Pennsylvania St., Indianapolis, Ind.
Waterman, Jas. S.....676 St. Marks Ave., Brooklyn, N. Y.
Webster, George A.....419 Boylston St., Boston, Mass.
Weil, Arthur I.....602 Title Guarantee Bldg., New Orleans, La.
Wells, Walter A.....The Rochambeau, Washington, D. C.
White, Joseph A.....200 East Franklin St., Richmond, Va.
Whiting, Frederick.....19 West 47th St., New York
Wiener, Alfred.....550 Park Ave., New York
Wilson, Harold.....1501 David Whitney Bldg., Detroit, Mich.
Wilson, Norton L.....410 Westminster Ave., Elizabeth, N. J.
Winslow, John R.....The Latrobe Apts., Charles & Read Sts., Baltimore, Md.
Wishart, D. J. Gibb.....45 Grosvenor St., Toronto, Can.
Wood, George B.....129 South 18th St., Philadelphia, Pa.
Woodward, John F.....153 Granby St., Norfolk, Va.
Worthington, Thos. C.....1022 Madison Ave., Baltimore, Md.
Yates, D. G.....149 West 81st St., New York
Yankauer, Sidney.....616 Madison Ave., New York

## HONORARY FELLOWS.

Ballance, C. E.....106 Harley St., London, W., Eng.
Bell, Alex. Graham.....1331 Connecticut Ave., N. W., Washington, D. C.
Chiari, Ottokar.....Bellariatrasse 12, Vienna, Austria
Gradenigo, Gius.....Corso Vittorre Emmanuele 44, Turin, Italy
Grant, Dundas.....18 Cavendish Square, London, W., Eng.
Heryng, T.....Jasna 10, Warsaw, Poland, Russia
Jansen, A.....Hardenbergst 12, Berlin, Germany
Karwowski, C.....Warsaw, Poland, Russia
Killian, Gustav.....Berlin, Germany
Law, Edward.....8 Wimpole St., Cavendish Sq., London, Eng.
Love, James Kerr.....650 Shields Road, Pollokshields, Glasgow, Scotland.
Lue, H.....54 Rue de Varenne, Paris
MacIntyre, John.....179 Bath St., Glasgow, Scotland

Manasse, P.....Strassburg, Germany
Moure, E. J.....25 Cours du Jardin Public, Bordeaux, France
Onodi, Adolph.....B'Uetza 12, Budapesth, Hungary
Politzer, Adam.....1 Gongazagasse, Vienna
Semon, Sir Felix.....Rignal, Grest Missenden, Bucks, Eng.
Schmieglow, E.....18 Norregade, Copenhagen, Denmark
Sendziak, J.....10 Crywariska Str., Warsaw, Poland, Russia
Turner, Logan.....27 Walker Street, Edinburgh, Scotland
Ziem, C. H.....Danzig, Germany

## VICE-PRESIDENTS OF THE SOCIETY.

| | Eastern Section. | Southern Section. | Middle Section. | Western Section. |
|---|---|---|---|---|
| 1896 | Joseph O'Dwyer, M. D. | (Only one Vice-President elected.) | | |
| 1897 | F. L. Jack, M. D. | Wm. Sheppegrell, M. D. | J. S. Mahon, M. D. | J. E. Logan, M. D. |
| 1898 | A. G. Root, M. D. | A. W. Calhoun, M. D. | J. A. Thompson, M. D. | J. E. Schadle, M. D. |
| 1899 | C W. Richardson, M. D. | C. A. Thigpen, M. D. | Max Thorner, M. D. | H. L. Wagner, M. D. |
| 1900 | G. H. Makuen, M. D. | J. A. Stucky, M. D. | H. S. Straight, M. D. | Robert Levy, M. D. |
| 1901 | W. S. Renner, M. D. | J. A. White, M. D. | N. H. Pierce, M. D. | J. O. McReynolds, M. D. |
| 1902 | H. H. Curtis, M. D. | C. F. McGahan, M. D. | H. W. Loeb, M. D. | E. W. Fleming, M. D. |
| 1903 | F. C. Cobb, M. D. | Dunbar Roy, M. D. | L. C. Cline, M. D. | P. F. Gildea, M. D. |
| 1904 | G. L. Richards, M. D. | J. F. Woodward, M. D. | C. Jackson, M. D. | R. W. Payne, M. D. |
| 1905 | S. MacCuen Smith, M. D. | Wm. Cheatham, M. D. | Thomas Hubbard, M. D. | M. A. Goldstein, M. D. |
| 1906 | T. H. Halsted, M. D. | H. L. Meyers, M. D. | W. L. Ballenger, M. D. | H. B. Ellis, M. D. |
| 1907 | F. B. Sprague, M. D. | J. M. Ray, M. D. | J. M. Ingersoll, M. D. | W. C. Bane, M. D. |
| 1908 | E. A. Crockett, M. D. | H. O. Reik, M. D. | J. F. Barnhill, M. D. | W. H. Roberts, M. D. |
| 1909 | F. R. Packard, M. D. | John Dunn, M. D. | R. B. Canfield, M. D. | J. C. Beck, M. D. |
| 1910 | J. F. McCaw, M. D. | O. A. M. McKimmie, M. D. | H. J. Hartz, M. D. | A. R. Solenberger, M. D. |
| 1911 | H. P. Mosher, M. D. | J. A. Kent, M. D. | La Fayette Page, M. D. | Wm. H. Dudley, M. D. |
| 1912 | Theodore Corwin, M. D. | Otto Joachim, M. D. | John A. Thompson, M. D. | Thomas J. Gallaher, M. D. |
| 1913 | George C. Stout, M. D. | J. W. Jervey, M. D. | Wm. E. Sauer, M. D. | Hill Hastings, M. D. |
| 1914 | Lewis A. Coffin, M. D. | F. M. Thigpen, M. D. | B. R. Shurley, M. D. | Lorenzo B. Lockard, M. D. |
| 1915 | Stephen H. Lutz, M. D. | Halstead S. Hedges, M. D. | John W. Murphy, M. D. | Frank R. Spencer, M. D. |
| 1916 | John B. Rae, M. D. | Robert C. Lynch, M. D. | William B. Chamberlin, M. D. | Thomas E. Carmody, M. D. |
| 1917 | John F. Culp, M. D. | Richmond McKinney, M. D. | John E. Brown, M. D. | James A. Patterson, M. D. |
| 1918 | D. Crosby Greene, Jr., M. D. | Thomas W. Moore, M. D. | D. W. Layman, M. D. | H. B. Graham, M. D. |
| | | | | **Mid-Western Section.** |
| | | | | Frank R. Spencer, M. D. |

## SECRETARY AND TREASURER.

1896 to 1900
Robert Cunningham Myles, M. D.
1901 to 1906
Wendell C. Phillips, M. D.
1907 to 1916
Thomas J. Harris, M. D.
1917 William H. Haskin, M. D.

1901 to 1907
Ewing W. Day, M. D.
1908 Chevalier Jackson, M. D.
1909 to date.
Ewing W. Day, M. D.

## COUNCIL.

1896—T. P. Berens, M. D.
J. H. Billings, M. D.
D. L. Hubbard, M. D.
H. H. Curtis, M. D.
W. C. Phillips, M. D.
R. C. Myles, M. D.
1897—G. H. Makuen, M. D.
T. P. Berens, M. D.
W. C. Phillips, M. D.
H. H. Curtis, M. D.
E. W. Day, M. D.
D. L. Hubbard, M. D.
1898—J. E. Nichols, M. D.
H. H. Curtis, M. D.
L. A. Coffin, M. D.
W. C. Phillips, M. D.
E. W. Day, M. D.
C. W. Richardson, M. D.
S. E. Solly, M. D.
1899—W. H. Daly, M. D.
W. C. Phillips, M. D.
T. H. Halsted, M. D.
J. O. Roe, M. D.
H. W. Loeb, M. D.
T. C. Christy, M. D.
F. L. Jack, M. D.
D. L. Hubbard, M. D.
1900—S. E. Solly, M. D.
W. H. Daly, M. D.
A. W. Calhoun, M. D.
J. B. Clemens, M. D.
J. F. McKernon, M. D.
W. C. Phillips, M. D.
J. E. Sheppard, M. D.
J. Price Brown, M. D.
Max Thorner, M. D.
1901—D. B. Kyle, M. D.
S. MacCuen Smith, M. D.
S. E. Solly, M. D.
J. Price Brown, M. D.
W. H. Daly, M. D.
J. B. Clemens, M. D.
J. F. McKernon, M. D.
C. W. Richardson, M. D.
F. C. Cobb, M. D.
1902—D. B. Kyle, M. D.
S. E. Solly, M. D.
J. F. McKernon, M. D.
S. MacCuen Smith, M. D.
F. C. Cobb, M. D.
N. H. Pierce, M. D.
T. P. Berens, M. D.
J. A. White, M. D.
1903—C. W. Richardson, M. D.
R. C. Myles, M. D.
D. B. Kyle, M. D.
J. F. McKernon, M. D.
S. MacCuen Smith, M. D.
F. C. Cobb, M. D.
N. H. Pierce, M. D.
T. P. Berens, M. D.
C. R. Holmes, M. D.
1904—J. A. Stucky, M. D.
C. W. Richardson, M. D.
R. C. Myles, M. D.
A. G. Root, M. D.
T. P. Berens, M. D.
C. R. Holmes, M. D.
E. B. Dench, M. D.
F. L. Jack, M. D.
J. E. Schadle, M. D.
F. R. Packard, M. D.
1905—N. H. Pierce, M. D.
J. A. Stucky, M. D.
C. W. Richardson, M. D.
L. A. Coffin, M. D.
T. H. Halsted, M. D.
C. R. Holmes, M. D.
E. B. Dench, M. D.
F. L. Jack, M. D.
J. E. Schadle, M. D.
F. R. Packard, M. D.

1906—F. C. Cobb, M. D.
N. H. Pierce, M. D.
J. A. Stucky, M. D.
E. B. Dench, M. D.
F. L. Jack, M. D.
F. R. Packard, M. D.
L. A. Coffin, M. D.
T. H. Halsted, M. D.
J. F. McKernon, M. D.
H. W. Loeb, M. D.

1907—J. E. Logan, M. D.
F. C. Cobb, M. D.
N. H. Pierce, M. D.
L. A. Coffin, M. D.
T. H. Halsted, M. D.
J. F. McKernon, M. D.
H. W. Loeb, M. D.
C. W. Richardson, M. D.
D. B. Kyle, M. D.

1908—W. C. Phillips, M. D.
J. E. Logan, M. D.
F. C. Cobb, M. D.
J. F. McKernon, M. D.
H. W. Loeb, M. D.
C. W. Richardson, M. D.
D. B. Kyle, M. D.
C. R. Holmes, M. D.
J. M. McCaw, M. D.

1909—J. E. Logan, M. D.
D. B. Kyle, M. D.
C. W. Richardson, M. D.
W. C. Phillips, M. D.
J. F. McCaw, M. D.
W. R. Lincoln, M. D.
G. L. Richards, M. D.
J. F. Barnhill, M. D.
G. H. Makuen, M. D.

1910—C. R. Holmes, M. D.
G. L. Richards, M. D.
J. F. Barnhill, M. D.
G. H. Makuen, M. D.
N. H. Pierce, M. D.
W. C. Phillips, M. D.
F. R. Packard, M. D.
A. B. Duel, M. D.
Robert Levy, M. D.

1911—J. F. McKernon, M. D.
J. A. White, M. D.
N. L. Wilson, M. D.
C. R. Holmes, M. D.
A. B. Duel, M. D.
F. R. Packard, M. D.
Robert Levy, M. D.
G. L. Richards, M. D.
N. H. Pierce, M. D.

1912—C. R. Holmes, M. D.
F. R. Packard, M. D.
N. H. Pierce, M. D.
J. F. McKernon, M. D.
J. A. White, M. D.
N. L. Wilson, M. D.
C. Jackson, M. D.
M. A. Goldstein, M. D.
A. B. Duel, M. D.

1913—James F. McKernon, M. D.
Joseph A. White, M. D.
Norton L. Wilson, M. D.
Chevalier Jackson, M. D.
Max A. Goldstein, M. D.
Arthur B. Duel, M. D.
Harris P. Mosher, M. D.
E. Fletcher Ingals, M. D.
G. Hudson-Makuen, M. D.

1914—Chevalier Jackson, M. D.
Max A. Goldstein, M. D.
Arthur B. Duel, M. D.
Harris P. Mosher, M. D.
E. Fletcher Ingals, M. D.
G. Hudson-Makuen, M. D.
H. Holbrook Curtis, M. D.
John E. Sheppard, M. D.
Robert Levy, M. D.

1915—Joseph A. White, M. D.
Harris P. Mosher, M. D.
E. Fletcher Ingals, M. D.
G. Hudson-Makuen, M. D.
H. Holbrook Curtis, M. D.
John E. Sheppard, M. D.
Robert Milligan, M. D.
John M. Ingersoll, M. D.
B. R. Shurley, M. D.

1916—G. Hudson-Makuen, M. D.
H. Holbrook Curtis, M. D.
John E. Sheppard, M. D.
Robert Milligan, M. D.
John M. Ingersoll, M. D.
B. R. Shurley, M. D.
E. B. Dench, M. D.
D. J. Gibb Wishart, M. D.
Robert Levy, M. D.

1917—Robert Milligan, M. D.
John M. Ingersoll, M. D.
E. B. Dench, M. D.
D. J. Gibb Wishart, M. D.
B. R. Shurley, M. D.
Robert Levy, M. D.
S. MacCuen Smith, M. D.
Thomas H. Halsted, M. D.
Walter A. Wells, M. D.

1918—D. J. Gibb Wishart, M. D.
B. R. Shurly, M. D.
Robert Levy, M. D
S. MacCuen Smith, M. D.
Thomas H. Halsted, M. D.
Walter A. Wells, M. D.
T. J. Harris, M. D.
Francis P. Emerson, M. D.
L. W. Dean, M. D.

## DECEASED WHILE MEMBERS OF THE SOCIETY.

| Resident Members. | | Members. | |
|---|---|---|---|
| Noyes, J. I., M. D. | 1896 | Mullins, John B., M. D. | 1909 |
| O'Dwyer, Joseph, M. D. | 1897 | McGahan, C. F., M. D. | 1910 |
| Hengst, D. A., M. D. | 1898 | Anderson, Willis S., M. D. | 1910 |
| Nichols, J. E. H., M. D. | 1898 | Calhoun, A. W., M. D. | 1910 |
| Hopkins, Woolsey, M. D. | 1899 | Gradle, Henry, M. D. | 1911 |
| Rankin, D., M. D. | 1900 | Koyle, F. H., M. D. | 1911 |
| Wenner, R. J., M. D. | 1900 | Dudley, Willam F., M. D. | 1912 |
| Chapman, S. Hartwell, M. D. | 1903 | Kimball, Irving E., M. D. | 1913 |
| Friedenberg, Edward, M. D. | 1903 | Gildea, Patrick F., M. D. | 1913 |
| Kibbe, A. B., M. D. | 1903 | Adams, J. L. M. D. | 1914 |
| Hoople, H. Nelson, M. D. | 1905 | Gibb, Jos. S., M. D. | 1914 |
| Brandegee, Wm. P., M. D. | 1906 | Gruening, Emil, M. D. | 1914 |
| Solly, S. E., M. D. | 1906 | Sheppard, John E. | 1916 |
| Harland, W. G. B., M. D. | 1907 | Roe, John O. | 1916 |
| Burnett, Peter V., M. D. | 1908 | Ballenger, Wm. L. | 1916 |
| Roosa, D. B., St. John, M. D. | 1908 | Dye, Hobart S. | 1917 |
| Schadle, J. E., M. D. | 1908 | Kyle, D. Braden | 1917 |
| Pette, C. H., M. D. | | Maknen, G. Hudson | 1917 |
| Sprague, F. B., M. D. | 1909 | Solenberger, Amos R. | 1917 |

## HONORARY MEMBERS WHO HAVE DIED.

| | | | |
|---|---|---|---|
| Whistler, W. McNeil | 1899 | Smyly, Sir Philip | 1904 |
| Gougenheim, Achille | 1901 | Joal, Joseph | |
| Gruber, Joseph | 1901 | Woakes, Edward | |
| Brown, Lennox | 1902 | | |

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Rhinological and Otological
Society
Transactions

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